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OBSERVATIONS ON THE PATHOLOGY OF IMPAIRED HEARING FOR LOW TONES.*

DR. DAIKICHI ODA, Baltimore.†

INTRODUCTION.

This paper reports the histologic study of 35 ears which had impaired hearing, in most cases of marked degree, for all tones with frequencies above 1,024 d.v. per second and slight or moderate impairment of hearing for all the lower tones. In each case, the 512 d.v. tuning fork was heard longer by air conduction than by bone conduction; *i.e.*, the Rinne test was positive. The generally accepted interpretation of such clinical tests is that the impaired hearing for the low tones, as well as for the high tones, is caused by inner ear or nerve lesions. For the high tones, this interpretation was placed on a firm basis by the report of Crowe, Guild and Polvogt (1934) on 79 ears with good hearing for low tones and impaired hearing for high tones. With respect to the low tones, however, this interpretation is supported mostly by theoretical considerations, rather than by histopathologic evidence. In the present study, therefore, although all lesions found are recorded and discussed, the primary interest is in

*From the Otological Research Laboratory, The Johns Hopkins University.

†The author, on leave from his post in the Department of Otolaryngology of the Medical School of the University of Okayama, takes with pleasure this opportunity publicly to express to Dr. Crowe and Dr. Guild his great appreciation of the permission to study the material in the Otological Research of the Johns Hopkins University, and of the helpful suggestions and generous assistance they have given him at all stages of this investigation.

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the lesions that offer explanations for the impaired hearing for low tones. Only seven, or one-fifth, of the ears have lesions in the location to be expected from classical theory; *i.e.*, atrophies in the middle or apical turns of the cochlea.

MATERIAL AND METHOD OF STUDY.

The methods of examining the hearing, of securing the temporal bones and of preparing them for histologic study are described in the paper by Crowe, Guild and Polvogt. For the present study the material in the laboratory's collection which meets the following requirements has been used:

1. The audiogram shows impairment of more than 20 dcb. for all frequencies, and the tuning fork tests show air conduction better than bone conduction. In most cases, the impairment for high tones is greater than for low tones.

2. The histologic preservation is good enough to permit judgment, whether or not the structures, especially those of the inner ear, were normal or pathologic during life.

Thirty-five ears meet the above requirements. In about two-thirds of these the time interval between examination of hearing and death was less than six weeks (from three to eight days for 11 of the ears, and from 11 to 19 days for eight of the others). The longest intervals were two and one-third years (two ears) and three years (one ear).

The 35 ears are from 19 individuals between the ages of 21 and 74 years; the average of the ages is 55 years. Two ears are from a woman, age 66 years, all the other material is from males.

The material has been studied and the observations recorded by the same general procedure employed by Crowe, *et al.* Briefly, they are as follows: For each cochlea the location and type of lesions found in the serial sections are recorded on graphic reconstructions (spiral graphs), then transferred to linear charts for ease of comparison of data from groups of cases. The method of doing this is described in detail in their paper, pages 320-337. The variation in total length of the organ of Corti in man is about 10 mm., from a minimum of 25.26 mm. to a maximum of 35.46 mm. (Hardy, 1938); therefore, in reducing all cases to a common length, as is necessary in the linear charts, there is for some ears a slight

loss of accuracy of location of lesions. The convenience afforded in the comparison of numerous records, however, more than compensates for the slight errors in exact extent of lesions.

The middle ear, for purposes of tabulation of observations, has been divided into nine regions. The manner of division and of recording conditions in each region are the same as in the paper by Crowe, *et al.*

OBSERVATIONS AND DISCUSSION.

Only rarely are all structures of the middle and the inner ear entirely normal; therefore, the lesions found in ears with impaired hearing must be more extensive or different than those in any ears with good hearing before they can be regarded as the cause of the hearing impairment. Furthermore, with respect to the special objective of the present study, it must be kept in mind that, to explain the impaired hearing for low tones, a lesion must either be more severe or in a different location than those in ears with impaired hearing for high tones only.

Therefore, the control groups for this investigation consist of:

I. Ears with good hearing for tones of all frequencies up to and including 8,192 d.v.

II. Ears with impaired hearing for high tones only.

CONTROL GROUP I.

This group consists of the same 15 ears with good hearing that were used as the control group by Crowe, *et al.*, in the paper on the pathology of high-tone deafness. For the present study it has been necessary to add to their records a detailed charting of the conditions in the middle and apical turns of the cochleae.

The data are recorded in Chart 1. Only the histologic observations require further elucidation.

The Cochlear Nerve: In all the regions of the cochlea except the lower part of the basal turn, the bundles of nerve fibres always fill the canals in the osseous spiral lamina. The lower part of the basal turn always has less nerve fibres than

the other parts of the cochlea, and in the last 4 mm. or so there is marked variation in the ears with good hearing. Often there is a partial atrophy averaging as much as 50 per cent in the basalward 4 mm., and 20 per cent in the next 3 mm.; therefore, atrophies of only this amount afford no explanation of impairment of hearing for either high or low tones.

The Organ of Corti: The extreme basal end of the organ of Corti is not always normal. In the last 2 mm., atrophy, partial or even total, is sometimes present. On the other hand, the organ of Corti is normal in structure at all higher levels; *i.e.*, in most of the basal and in all of the middle and apical turns.

The External Sulcus Cells: The penetration of the external sulcus cells into the spiral ligament is not well demonstrated by the ordinary stains used with this material; therefore, only the appearance of the superficial cells may be discussed. These normally consist of high columnal cells in the basal turn, and of low columnar or cuboidal cells in the middle and apical turns. In ears with good hearing, this epithelium frequently has atrophic changes in the last few millimeters of the basal turn. In other parts of the cochlea, the external sulcus cells are always normal in appearance.

The Stria Vascularis: The stria vascularis is regarded as normal in extent when it covers the spiral ligament from the base of the prominentia spiralis to the attachment of Reissner's membrane. In only one of the 15 ears of this control group does the stria vascularis cover the maximum surface at all levels of the cochlea. The defect is very noticeable in eight of these ears. Inspection of Chart 1 shows that on the average the strial defect is somewhat greater in the upper turns than in the basal turn.

The Middle Ear: The control group for the middle ear is larger than the 15 ears charted, thanks to Polvogt's report on lesions of the middle ear in 63 cases with good hearing for all frequencies up to and including 8,192 d.v. The following conditions may not cause any impairment of hearing: *1.* marginal or moderate diffuse thickening of the tympanic membrane; *2.* small scars and calcified areas in the tympanic membrane; *3.* retraction of Shrapnell's membrane without formation of much scar tissue in Prussak's space; *4.* fibrous

adhesion of the posterior crus of the stapes to the promontory; *5.* small exostoses near the anterior margin of the oval window, which do not impinge upon the footplate of the stapes or have fibrous adhesion to its anterior crus; *6.* fibrous bands or small masses of myxomatous or other tissue in the round-window niche; *7.* fibrous thickening of the mucosa of the promontorial region or of the hypotympanum; and *8.* thickenings of bone and mucosa in the anterosuperior part of the middle ear that do not, however, involve the orifice of the Eustachian tube or the ossicles.

CONTROL GROUP II.

This control group consists of the 79 ears with impaired hearing for high tones only, reported by Crowe, Guild and Polvogt. For details of their findings, the original publication must be consulted. Only the observations of most importance for the present study are summarized here.

The cochlear lesions more severe than in the ears with good hearing are limited to the basal turn. In about three-fourths of the 79 ears, the basal turn has atrophy of nerve, or of nerve and organ of Corti, more extensive and severe than in any ears of their control group. For the ears with audiograms of a "gradual" high-tone loss type, the most striking lesion is the partial atrophy of nerve; there is more nerve in the upper than in the lower part of the basal turn, and it is always normal in amount in the middle and apical turns. When the audiogram shows an "abrupt" high-tone loss, there is, with few exceptions, atrophy of the organ of Corti, as well as of the nerve; the atrophy of the end-organ is usually limited to part or all of the lower basal turn and is associated with atrophy of external sulcus cells of corresponding or of somewhat greater extent. Strial defects more extensive than in the ears with good hearing were but seldom found, and in no case did these authors regard the impaired hearing as due to strial lesions. Their observations provide positive evidence as to localization for the frequencies of 2,048, 4,096 and 8,192 d.v. The regions of most importance for these frequencies are located, respectively, in the lower part of the upper basal turn, in the upper part of the lower basal turn, and about the middle of the lower basal turn.

Seven of the 79 ears have middle-ear lesions that are not present in cases with good hearing for all tones. The lesions

are fully described in the original paper. All seven of these ears also have cochlear lesions, regarded by the authors as sufficient to explain the impaired hearing for high tones. For this reason, no conclusions could be drawn as to the effect of the middle-ear lesions upon hearing for high tones. It is certainly surprising that they did not cause impairment of hearing for low tones.

With the important facts of the two control groups in mind, the data for the 35 ears with impaired hearing for low, as well as for high, tones can be more intelligently analyzed. The presentation is divided into two parts: *1.* description of the histologic lesions observed; and *2.* correlations between clinical tests and structural lesions.

1. THE LESIONS IN 35 EARS WITH IMPAIRED HEARING FOR LOW AND FOR HIGH TONES.

On the basis of the histologic findings, especially as to whether or not there are lesions in the middle and apical turns, or in the middle ear, beyond normal limits, the 35 ears have been divided into four groups. The data are given in Chart 2. Within each group the ears have been arranged in order of the average of the thresholds of hearing for the tones of c, $c,^1$ c^2 and c^3 (128 to 1,024 d.v., inclusive).

A. This group includes 16 ears. The cochlear lesions, except for stria vascularis, are limited to the basal turn. The middle-ear lesions are within the limits of Control Group I.

B. The cochleae of these five ears are like those of the preceding group. The middle-ear lesions are rather marked but may possibly be within normal limits.

C. The seven ears of this group have lesions of the middle ear definitely more severe than seen in ears with normal hearing. Cochlear lesions are as in the preceding groups.

D. These seven ears are grouped together because in each the cochlear lesions are definitely more extensive than in any ears of Control Group II. The atrophic changes in the nerve extend into the middle and apical turns.

The lesions of the stria vascularis, the external sulcus cells and the basilar membrane are similar in the four groups. To avoid repetition of descriptions, consideration of these struc-

tures is grouped together. The other structures are considered separately under the heading of each group.

The Stria Vascularis: The nature of the strial defect is described under this heading in Control Group I. Comparison of Chart 1 with Chart 2, with respect to the data for the stria vascularis, shows that in each of the four groups of Chart 2 the range of variation in ears with impaired hearing is about the same as in ears with good hearing.

The External Sulcus Cells: The association of atrophy of the external sulcus cells with atrophy of the adjacent part of the organ of Corti is similar to that found in Control Group II. In both groups, when there is a difference, the atrophy of external sulcus cells extends over a longer area than does the atrophy of the end-organ. Crowe and his associates suggested that there may be a nutritional relationship between the two structures, and that the atrophy of the external sulcus cells of a region precedes atrophy of the organ of Corti. This interpretation is in agreement with the present observations.

In only one ear does the atrophy of the external sulcus cells extend beyond the limits of ears of Control Group II. Other lesions in this ear afford sufficient explanation for the impaired hearing. Obviously, lesions of the external sulcus cells cannot be regarded as the direct cause of impaired hearing for low tones.

The Basilar Membrane: The histologic appearances termed hyalinization and calcification (Mayer, 1919) have been judged and recorded in the same way as by Crowe, *et al.* In two-thirds of the 35 ears of Chart 2, such changes are more extensive than in any of Control Group I (Chart 1). At most, however, they do not extend beyond the middle of the lower basal turn and cannot be regarded as responsible in any ear for the impaired hearing for low tones.

GROUP A.

The 16 ears (Nos. 1 to 16, Chart 2) are from 10 patients between 44 and 66 years of age. One was a female. Four of them were younger than 50 years, only one was older than 60 years.

The Middle Ear: The lesions seen in these ears which might interfere somewhat with ossicular movements are:

1. A small otosclerotic focus in the central part of the footplate of the stapes in one ear (No. 1).

2. Eight ears (Nos. 2, 3, 4, 5, 7, 8, 14 and 16) have a narrow or a more or less wide band of loose myxomatous tissue between the anterior or the posterior crus (mostly posterior) of the stapes and the promontory or the eminentia canalis facialis, or both of them.

3. Myxomatous tissue partially fills Prussak's space, and is also present between the posterior crus of the stapes and the promontory, in two ears (Nos. 11 and 12).

In three ears of this group (Nos. 2, 9 and 13) the malleus, incus, epitympanum and pars flaccida of the tympanic membrane were destroyed at autopsy. No lesions beyond normal limits are seen in other parts of these middle ears.

The annular ligament of ear No. 1, which has the otosclerotic focus in the stapes, is not involved. All the other lesions mentioned above are definitely within the range of the pathologic changes occasionally seen in ears with good hearing.

The Inner Ear: Eleven ears (Nos. 2, 3, 4, 5, 6, 9, 10, 11, 12, 15 and 16) have atrophy of the nerve and of the organ of Corti beyond the limits of Control Group I; in none do the lesions extend beyond the basal turn. The amount of nerve atrophy in the upper basal turn is slight, compared to that in the lower basal turn. In three of these 11 ears, the partial atrophy of the nerve is limited to the lower basal turn; only in five does it extend, even in slight degree, into the upper half of the upper basal turn. The end-organ lesions vary from an extensive area of total atrophy, as in ear No. 9, to absence of one or more hair cells in some sections. Three ears (Nos. 4, 5 and 15) have one or several small areas with hair cells missing in the upper basal turn; otherwise, the lesions of the organ of Corti are limited to the lower basal turn.

In all 11 of these ears, the hearing for high tones was much worse than for the low tones. The hearing decrease, from the general level for low tones, occurred "abruptly" in seven ears (Nos. 2, 3, 9, 10, 11, 12 and 15), and "gradually" in four ears (Nos. 4, 5, 6 and 16). Comparison with Control Group II shows that without doubt the cochlear lesions in each of these 11 ears are sufficient to explain the impairment of hearing for high tones. Consideration of whether or not the lesions

afford any explanation for the impairment for low tones will be postponed until the lesions of the other groups are described.

Of the remaining five ears of Group A, one (No. 13) has a partial atrophy of the nerve in the lower basal turn, but no lesion of the organ of Corti; the audiogram shows a "gradual" type of impairment for successively higher tones. The other four ears (Nos. 1, 7, 8 and 14) have no cochlear lesions more severe than in Control Group I; *i.e.*, no explanation even for the impaired hearing for high tones.

GROUP B.

The five ears of this group (Nos. 17 to 21, Chart 2) are from three males, age 21, 51 and 59 years.

The middle-ear lesions are within the limits described by Polvogt for ears with good hearing, but each ear of this group has a lesion more severe than is commonly seen in the control group. Two ears (Nos. 17 and 18) have a dense fibrous adhesion of the capitulum of the malleus to a small bony process of the tegmen tympani. The other three ears (Nos. 19, 20 and 21) have a very wide adhesion between the posterior crus of the stapes and the promontory.

The cochlear lesions, as in Group A, are limited to the basal turn; in all five ears they are more severe than in the ears of Control Group I. In four, there is atrophy of both the organ of Corti and the nerve; in one (No. 19), the organ of Corti is intact at all levels.

The most extensive lesion is in ear No. 21, in which the atrophy of nerve, of organ of Corti and of external sulcus cells is continuous from the basal end of the cochlea into the beginning of the upper basal turn. The condition is similar to that of ear No. 70 of Control Group II (see Chart 5 of paper by Crowe, *et al.*). Both ears had an "abrupt" drop of 50 dcb. in the threshold for 2,048 d.v., compared to that for 1,024 d.v. It is quite possible that the effect of the middle-ear lesion in the present case (No. 21), when added to that of the cochlear lesions, is responsible for the difference in hearing for low tones by the two ears. In the same way, it is possible that the combined effect of middle-ear lesions and cochlear lesions in the other four ears of this group explain

the impaired hearing for low tones, even though neither lesion by itself causes such an impairment.

GROUP C.

The seven ears of this group (Nos. 22 to 28, Chart 2) are from six individuals, all males, whose ages were 21, 44, 47, 52, 53 and 74 years. The ears are grouped together because in each there is a middle-ear lesion definitely more severe than has been seen in any ears with good hearing.

Both ears of the man, age 74 years (Nos. 22 and 23), have dense fibrous adhesions of the anterior crus of the stapes and the adjacent part of the footplate to the anterior margin of the oval window and the nearby part of the promontory. These adhesions are associated with an otosclerotic process histologically quiescent, which has not caused any bony ankylosis. In spite of this condition, both ears heard the 512 d.v. tuning fork definitely longer by air conduction than by bone conduction. Until the histologic sections were prepared the presence of otosclerosis was not suspected. In both cochleae there is a partial atrophy of nerve in the lower basal turn sufficient to explain the impaired hearing for high tones.

Ear No. 27 also has histologic otosclerosis. The process is limited to the footplate of the stapes. Near the centre of the inferior margin, the articular cartilage and the annular ligament have been replaced by the otosclerotic formation, so that here the footplate is in direct contact with the margin of the oval window but is not ankylosed to it. The cochlear lesions are within the limits of ears with good hearing.

In another ear of this group (No. 26), the anterior part of the footplate of the stapes is thicker than normal and is bound to the promontory near the anteroinferior part of the oval window by fibrous tissue. The change in the footplate may be due to otosclerosis, but more probably was caused by a simple osteitis. Also in this ear, the space between the pars flaccida of the tympanic membrane and the neck of the malleus is filled with dense fibrous tissue. There are also changes obviously due to an acute otitis media, which developed shortly before death; these need not be considered in the interpretation of the functional tests made three months previously. In this case, the 512 d.v. tuning fork was heard but slightly better by air conduction than by bone conduction. There is

partial atrophy of the nerve in the lower basal turn of the cochlea, but not more than is seen in many ears with impaired hearing for high tones only.

Ear No. 25 has a dense fibrous, partially calcified adhesion of the capitulum of the malleus to a small bony process of the tegmen tympani, and a fibrous adhesion of the inferior surface of the anterior stapedial crus to the promontory near the oval-window margin. The promontory, in the region of attachment of the adhesion, is thickened, apparently because of an osteitis now healed. In the basal 8 mm. of the cochlea there is total atrophy of the nerve, the organ of Corti and the external sulcus cells. Partial atrophy of the nerve extends into the beginning of the upper basal turn. The cochlear lesions account fully for the total loss of hearing for the frequencies of 4,096 and 8,192 d.v.

One ear (No. 28) has a large mass of myxomatous, cystic tissue in the upper part of the tympanic cavity; it is attached to all the ossicles and partially fills the fossula of the oval window. Prussak's space is also filled with the same kind of tissue. The slight signs of acute inflammation, infiltration of leukocytes and round cells, probably developed after the hearing tests made two months before death. The cochlear lesions are within the limits of Control Group I.

The tympanic membrane of ear No. 24 has a marked degree of fibrous thickening and calcification of the anterior half, and a very large thin atrophic area in all but the marginal region of the inferior and posterior parts. Prussak's space is partially filled with dense fibrous tissue. The cochlea has a partial atrophy of the nerve in the lower part of the basal turn, slightly beyond the limits of ears with good hearing.

GROUP D.

The seven ears (Nos. 29 to 35, Chart 2) of this group are from four individuals, all males. Two were age 63 years, the others were age 60 and 73 years. Except for one person (ears Nos. 30 and 31), the interval between the hearing test and death was very short (15, eight and four days, respectively). This is of especial importance, in view of the fact that this is the group in which the impaired hearing for low tones is best explained by the cochlear lesions of the middle and apical turns.

There are no lesions of middle-ear structures beyond normal limits in any ear of this group. Both ears of one man (Nos. 32 and 34) have otosclerotic foci in the lateral margin of the round-window niche; in addition, the right ear has a focus in the anterior margin of the oval window. But there is no ankylosis of the stapes. This change could not have interfered with the transmission of sound.

All ears of this group have extensive partial atrophy of the nerve. In some, it is continuous (Nos. 32, 34 and 35); in others, it is more or less localized (Nos. 29, 30, 31 and 33);

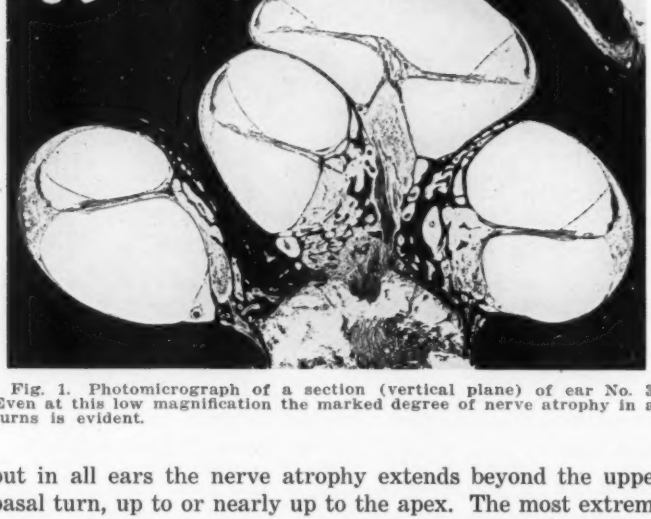

Fig. 1. Photomicrograph of a section (vertical plane) of ear No. 34. Even at this low magnification the marked degree of nerve atrophy in all turns is evident.

but in all ears the nerve atrophy extends beyond the upper basal turn, up to or nearly up to the apex. The most extreme degree of nerve atrophy is in ear No. 34, a photomicrograph of which is reproduced in Fig. 1. The average age for this group is over 10 years more than for any of the other groups. None of the ears in Group D are from patients less than 60 years of age. The number of cases is too small, however, to warrant the conclusion that the age-factor is responsible for the greater extent of the nerve atrophy.

In each ear of this group, the extent of the nerve atrophy greatly exceeds that of the organ of Corti. The condition of

the organ of Corti in two ears (Nos. 30 and 31) is within the limits of Control Group I. In four ears (Nos. 29, 32, 33 and 34), the atrophy is limited to the lower basal turn, as in ears with impaired hearing for high tones only. Only in ear No. 35 does the extent of the end-organ atrophy exceed that in any ears of Control Group II. This cochlea, obtained only four days after the hearing was examined, has total atrophy of the organ of Corti the entire length of the basal

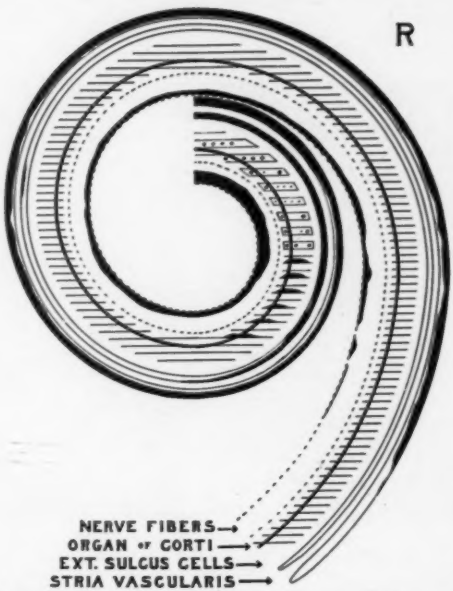

Fig. 2. This graphic reconstruction of ear No. 35 shows the extensive atrophy of end-organ and nerve in the basal and the lower middle turns. In all parts of the upper middle and the apical turns, not shown in this reconstruction, the organ of Corti is normal, but there is partial atrophy of the nerve. (See Fig. 3 and Chart 2.)

turn, or for about 17 mm. from the basal end. In addition, for the first 2.5 mm. of the lower middle turn there is an "almost total atrophy," such as illustrated in Fig. 5 of the paper by Crowe, *et al.;* and for the next 2 mm. some hair cells are missing in part of the sections. Only above the 21.5 mm. level, as measured from the basal end, is the organ of Corti histologically normal. The spiral chart of this cochlea is shown in Fig. 2, and a photomicrograph of a midmodiolar section in Fig. 3.

2. Correlations Between Clinical Tests and Structural Lesions.

Both the "gradual" and the "abrupt" types of high-tone impairment, into which Crowe, Guild and Polvogt divided their material, may be recognized in the audiograms of the present material. The position on the chart of the thresholds for low tones is, of course, different, but the audiograms have the same two types of transition from hearing acuity for low tones to that for high tones. There is also a third type of audiogram in the present material, which is best described

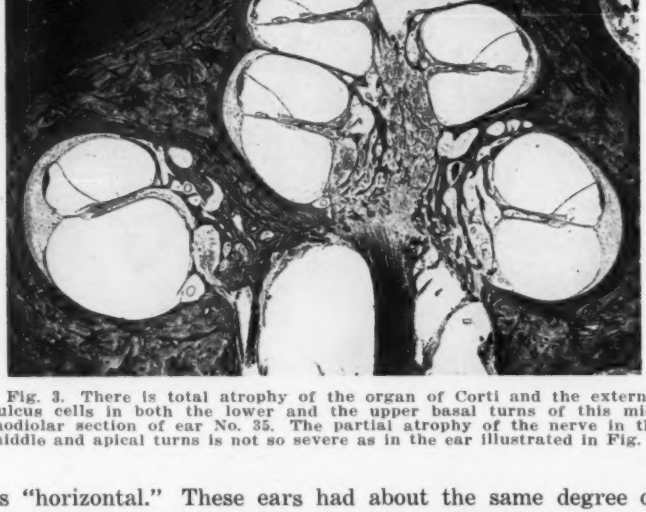

Fig. 3. There is total atrophy of the organ of Corti and the external sulcus cells in both the lower and the upper basal turns of this midmodiolar section of ear No. 35. The partial atrophy of the nerve in the middle and apical turns is not so severe as in the ear illustrated in Fig. 1.

as "horizontal." These ears had about the same degree of impairment for high and for low tones. Examples of these three types of audiograms are shown in Fig. 4.

The seven ears of Group D have lesions in good agreement with the usual theoretical interpretations of such functional tests. Six of these ears have a gradual decrease for the successively higher tones; in two (Nos. 30 and 31) of the six, this decrease is somewhat irregular, but in the other four (Nos. 29, 32, 33 and 34) it is typical. In all six ears the extent of the lesion of the organ of Corti is small in compari-

son to that of the nerve, as may be seen by inspection of their records in Chart 2. On the other hand, ear No. 35, in which the end-organ atrophy is continuous from the basal end into the lower middle turn (see Figs. 2 and 3), has an "abrupt"

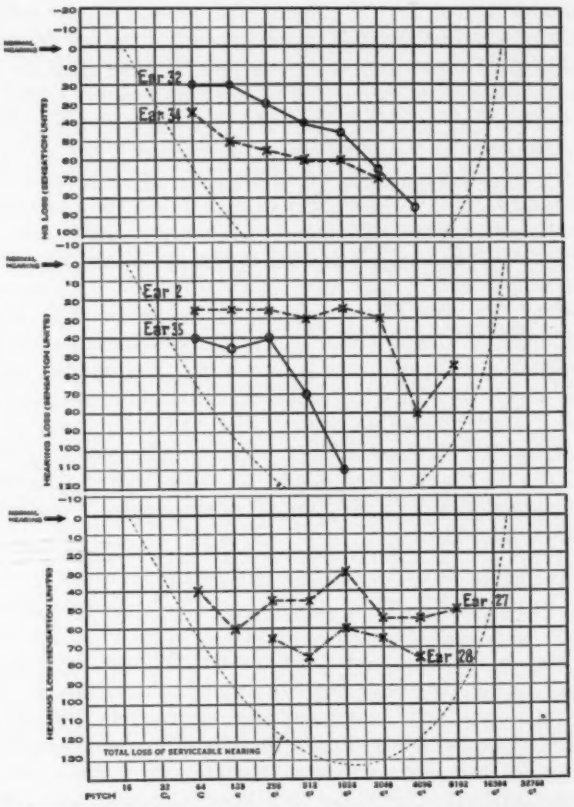

Fig. 4. Two examples of each of the three types of audiograms. The first two ears had a "gradual" decline for successively higher tones and the next two had an "abrupt" drop in acuity of hearing for high tones. The lower two audiograms are classified as "horizontal."

decrease for higher tones that begins at 512 d.v. (see Fig. 4). The relations of the types of hearing impairment for higher tones to the two types of lesions remind one of the observations of Crowe, Guild and Polvogt. The difference in hearing

acuity for low tones, in their cases and in the ears of Group D of the present material, is easily accounted for by the difference in the amount of nerve in the middle and apical turns. Their cases had good hearing for low tones and a normal amount of nerve in these turns.

Comparison of ear No. 34 of the present study with ear No. 55 of Control Group II (see Chart 5 of paper by Crowe, *et al.*) illustrates well the correlations that can be made between functional tests and structural lesions. Both ears had total loss of hearing for 4,096 and 8,192 d.v.; ear No. 55 had good hearing for all tones up to and including 2,048 d.v., but ear No. 34 had impaired hearing for all frequencies that were heard, with a "gradual" decline from the threshold of 35 dcb. for 64 d.v. to 70 dcb. for 2,048 d.v. Both ears have atrophy of organ of Corti and external sulcus cells almost to the upper end of the lower basal turn. In ear No. 55 the extent of the area of nerve atrophy is the same as that of the end-organ atrophy, but in ear No. 34 the nerve atrophy extends on up through the upper basal, the middle and the apical turns (see Chart 2 and Fig. 1). This extensive nerve atrophy explains satisfactorily why the audiogram of ear No. 34 has a "gradual" rather than an "abrupt" drop to the total loss of hearing for 4,096 d.v. and higher frequencies always associated with the amount of end-organ atrophy present in its basal turn.

In the same way, comparison of ear No. 34 with ear No. 9 of the present material is instructive. The end-organ atrophy in both is of about the same extent and accounts fully for the total loss of hearing for 4,096 and 8,192 d.v. The partial atrophy of nerve in ear No. 34 extends to the apex, but in ear No. 9 only into the upper basal turn. This difference in the lesions correlates nicely with the difference in acuity of hearing for the tones below a frequency of 4,096 d.v. — the audiograms up to 2,048 d.v. are approximately parallel and from 15 to 25 dcb. apart.

When the entire material of 35 ears is grouped on the basis of the three types of audiograms described above, 14 fall into the classification of "abrupt" drop for high tones, 15 into the "gradual" decrease group, and six into the "horizontal" group. Subdivision of these groups on the basis of the lesions reveals some points of interest.

"Abrupt" Loss Group: Thirteen* of the 14 ears with this type of audiogram have atrophy of the organ of Corti more extensive than in Control Group I. Since this is the characteristic lesion of those ears in Control Group II that had an "abrupt" loss type of audiogram, there can be no doubt that, when there is also some impairment for low tones, the end-organ atrophy is responsible for the marked impairment of hearing for high tones. This conclusion is strengthened by the fact that, in all but one ear (No. 35), the "abrupt" drop in hearing acuity occurs above 1,024 or 2,048 d.v., just as in the ears with good hearing for low tones.

Therefore, in the correlation of lesions and hearing tests of this group, only the question remains: How explain the impairment for low tones?

On the average, the ears of Control Group II that have good hearing for low tones and an "abrupt" loss for high tones have either less nerve atrophy than in the present material or less severe middle-ear lesions. In only one of the 13 ears now under consideration is the nerve atrophy confined to the lower basal turn; also, in only one ear (No. 35, described above in detail, does the nerve atrophy extend the entire length of the cochlea. For most of the ears the difference in condition of the nerve is in the upper basal turn. The evidence is not conclusive but does suggest that possibly the upper basal turn of the cochlea has some part in the hearing of low tones.

Five of these 13 ears also have middle-ear lesions that may be wholly or in part responsible for the impairment for low tones. In three of the five (Nos. 18, 20 and 21, of Group B), these lesions are not more severe than sometimes found in ears with good hearing for all tones, but, as has been mentioned above, it is quite possible that when there is also a cochlear lesion the combined effect is to cause impairment for low tones. In the other two ears of the five (Nos. 23 and 25, of Group C), the middle-ear lesions are definitely beyond the limits of Control Group I.

Of the 14 ears with audiograms of the "abrupt" drop type, only one (No. 8) has no lesion of cochlea or of middle ear beyond the limits of those frequently found in ears with good hearing.

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*These are Nos. 2, 3, 9, 10, 11, 12, 15, 18, 20, 21, 23, 25 and 35.

"Gradual" Decrease Group: For six of the 15 ears of this group, extensive nerve atrophy is the prominent lesion and there is perfect correlation, along the lines of classical theory, between the functional tests and the cochlear lesions. These are ears Nos. 29 to 34, inclusive, of Chart 2. All have partial atrophy of the nerve in all turns of the cochlea. In the "gradual" high-tone loss cases of Control Group II (see Chart 2 of the paper by Crowe, *et al.*), the nerve atrophy is limited to the basal turn; therefore, the difference in hearing for low tones correlates well with the difference in extent of lesion in the two groups. The special correlations that can be made in the case of one of these six ears (No. 34) have already been considered (see page 780).

In two ears (Nos. 13 and 26), the cochlear lesions beyond those of ears with good hearing consist only of partial atrophy of the nerve in the lower basal turn. This accounts only for the impairment for high tones. One of them (No. 26) has a middle-ear lesion more severe than in Control Group I. No lesion has been found in the other ear that might explain the impaired hearing for low tones.

Four of the 15 ears of this group (Nos. 4, 5, 6 and 16) have lesions of both nerve and end-organ, limited to the basal turn, as in ears of Control Group II with an "abrupt" drop type of audiogram. None of the four ears have middle-ear lesions. The cochlear lesions account for the impaired hearing for high tones, but no explanation has been found either for the "gradual" type of the impairment or for the fact that low tones were not well heard.

Of the 15 ears of this "gradual" decrease group, three (Nos. 1, 7 and 14) have no lesions that explain even the impairment for high tones. So far as the limitations of the histologic technique permit judgment, the minor lesions in all three of these ears are well within the limits of Control Group I.

It is of interest to note, at this time, that in both the present study and that by Crowe, *et al.*, there are more "unexplained" impairments for high tones in ears with a "gradual" loss type of audiogram than in those with an "abrupt" loss. No explanation for this fact can be advanced at the present time. Certainly, however, the fact should stimulate future investigators to apply additional techniques to the histologic study of ears with a "gradual" transition in acuity of hearing from low to high tones.

"Horizontal" Audiogram Group: The prominent lesions in all six of the ears with this type of audiogram are those of the middle ear. Four of the six ears have middle-ear lesions definitely more severe than are found in ears with good hearing for all tones; the lesions are described above in the text concerning Group C. The cochlear lesions in two of these four ears (Nos. 27 and 28) are well within the limits of Control Group I; the other two ears (Nos. 22 and 24) have somewhat more atrophy of the nerve in the lower basal turn than is found in ears with good hearing.

The other two ears with this type of audiogram are Nos. 17 and 19, of Group B. The middle-ear lesions of both, described under the section heading, are similar to some observed by Polvogt in ears with good hearing for all tones. In his cases, the cochleae were histologically normal, but both of the present cases have some nerve atrophy in the basal turn and one also has areas in which part of the hair cells of the organ of Corti are missing.

Definite conclusions are not warranted by the evidence. However, the fact that all six of the ears with "horizontal" type audiograms have prominent middle-ear lesions would seem to be more than a mere coincidence.

LOCALIZATION.

Evidence as to tonal localization along the basilar membrane is provided by the correlations between hearing and lesions for certain of these ears. The best evidence is that from the ears with extensive atrophy of the organ of Corti and total loss of hearing for certain tones. The correlations possible when the organ of Corti is present are not so convincing or reliable. Retzius (1892), Lorente de Nó (1933) and others have demonstrated that nonmedullated fibres may course for a considerable distance, possibly a third of a turn or more, within the organ of Corti itself; therefore, the amount of nerve in the immediately adjacent part of the osseous spiral lamina is not necessarily a correct measure of the innervation of that part of the end-organ.

The special correlations as to localization for ear No. 34 have already been discussed (see page 780). Omitting for the moment ear No. 35, there are three other ears (Nos. 9, 21 and 25) with a total loss of hearing for both 4,096 and

8,192 d.v. In all three there is extensive atrophy of the organ of Corti; in Nos. 9 and 25 the area of atrophy extends almost to the upper end of the lower basal turn (see Chart 2) and in No. 21 into the beginning of the upper basal turn. For the latter ear, the threshold of the highest of the octave-interval tones heard, 2,048 d.v., was much more impaired than for the other two ears.* Thus, the evidence from these four ears (Nos. 9, 21, 25 and 34) is in complete agreement with the conclusions of Crowe, Guild and Polvogt as to the regions of the cochlea of most importance in the hearing of the frequencies of 4,096 and 2,048 d.v.

In the material of the previous study there were no ears with a total loss of hearing for 2,048 d.v. The present material contains but one example, ear No. 35. The fact that this ear also has more extensive end-organ atrophy than in any other ear of either study warrants a careful analysis of the data with respect to localization, even though conclusions based on only one case are necessarily tentative.

This ear had not only total loss of hearing for 2,048 d.v. and all higher frequencies but also an extreme impairment for the next lower octave, 1,024 d.v. (threshold at 110 dcb. above normal with the other ear masked). The frequency of 512 d.v., with a threshold of 70 dcb. above normal, is in the middle of the "abrupt" decline in hearing acuity from the general level of 40 dcb. for the lower frequencies (see Fig. 4). The atrophy of the organ of Corti in this ear (see Figs. 2 and 3 and Chart 2) is total in the entire basal turn and "almost total" in the lower 2.5 mm. of the middle turn. The next 2 mm. is a zone of transition, and only for the apicalward 10 mm. or so of the basilar membrane is the organ of Corti normal. Partial atrophy of the nerve extends all the way to the apical end. The total loss of hearing for 2,048 d.v. is in good agreement with the total atrophy of the end-organ in the region indicated by the other evidence as to localization for this tone — the lower part of the upper basal turn. It is with respect to the question of localization along the basilar membrane for the frequencies below 2,048 d.v., however, that this case is of special interest.

According to the "spacing" indicated by the location of the areas for 8,192, 4,096 and 2,048 d.v., respectively, the area

*The difference in impairment for low tones in these three ears is probably caused by the difference in the middle-ear, rather than in the cochlear, lesions.

for the frequency an octave lower, 1,024 d.v., would be in the upper part of the upper basal turn; *i.e.*, within the region of total atrophy of the organ of Corti in this ear. The fact that this frequency was heard at all only four days before death is good evidence that, even if there is some degree of localization in this part of the cochlea for 1,024 d.v., at high intensities the stimulus must be spread over a rather wide area of the basilar membrane. A similar assumption is necessary to explain the impairment for 512 d.v., since, on the usual theory of equal distances between octave tones the location for this frequency falls in the area of "almost total" atrophy of the end-organ.* The facts of this case do not agree at all with the theoretical locations assigned to these frequencies (1,024 and 512 d.v.) by Stevens, Davis and Lurie (1935), also by Stevens and Davis in their very recent book (May, 1938). Their schema shows the frequency of 1,000 d.v. about the junction of the lower and upper parts of the middle turn and the frequency of 500 d.v. in the lower end of the apical turn. On the basis of this theory, the thresholds for the ear under discussion should have been about equally impaired for all frequencies up to and including 1,024 d.v., instead of only up to 256 d.v.

The impairment of about the same amount, 40 dcb., for all frequencies up to and including 256 d.v. correlates well, along the lines of classical theory, with the normal end-organ and partial atrophy of nerve in the apical 10 mm. of the cochlea, and suggests that reception for the frequency of 256 d.v. is located somewhat more than 21.5 mm. from the basal end of the basilar membrane. In this connection, however, it must be kept in mind that there is no evidence from the study of human material to contradict the view that low tones can be heard whenever the organ of Corti of any region is intact and innervated (Guild, 1935; Crowe, 1938).

SUMMARY AND CONCLUSIONS.

Serial sections of 35 ears with impaired hearing for both low and high tones, and with the Rinne test positive, have been studied. Explanations of the poor hearing for low tones

*It is of interest to note that the animal experiments of Wittmaack (1907), Yoshii (1909) and others with acoustic trauma support the idea that intense stimuli spread over a considerable area of the basilar membrane. Exposure of their animals to loud tones for a long time caused degenerative changes of the end-organ over an area of a third to a half of a turn of the cochlea.

have been especially sought for. The interpretation of lesions possibly responsible for the impairment of thresholds for low tones is controlled from two angles: *1.* the range of lesions in 15 ears with good hearing for all tones; and *2.* the lesions found by Crowe, Guild and Polvogt in 79 ears with impaired hearing for high tones only. The sections of many of the ears of the latter group have also been studied by the writer in the course of the present investigation. The general method of study is the same as was employed by these authors. The ears are from 19 patients, only one of whom was female. The average age is 55 years; the youngest was 21 years, the oldest 74 years.

Complete data for the ears of Control Group I and for the 35 ears with impaired hearing for all tones are given in Charts 1 and 2, respectively. For Control Group II, ears with impaired hearing for high tones only, a summary of the observations by Crowe and his associates is given; the details may be found in the original article.

The 35 ears are divided into four groups on the basis of differences in severity and extent of lesions of the inner and of the middle ear, compared to those in the two control groups, and into three groups on the basis of types of audiograms. Correlations of functional performance (hearing tests) and structural conditions (lesions) are made by comparing one ear with another and the groups of ears with each other and with the control groups.

The cochlear lesions consist of various degrees of atrophy of one or more of the following structures: nerve, organ of Corti, external sulcus cells and stria vascularis. The histologic appearance is always that of a pure atrophy, without signs of a present or of a past inflammation. In no case is there cellular infiltration of the nerve or an increase in fibrous tissue. The differences in cochlear lesions are in the degree of atrophy and in the extent and location of the area involved.

Most of the lesions of the middle ear in this material are one or the other of three types: *1.* an increase of fibrous tissue in some location; *2.* masses or bands of myxomatous tissue; *3.* an otosclerotic area without ankylosis of the stapes.

No correlation has been found between the systemic diseases which the patients had and either the cochlear or the

middle-ear lesions. Certainly, syphilis is not the cause of the impaired hearing in these ears; only one of the 19 patients had this disease and there is nothing peculiar to his cochlear lesions.

A history of tinnitus, in no case of severe degree, was given by only five of the 19 patients; each referred it definitely to one ear.

Twenty-nine of the 35 ears had markedly more impairment of hearing for high tones than for low tones. On the basis of the audiograms, these are divided into two groups: *1.* those with about the same impairment for all tones up to some frequency, usually 1,024 or 2,048 d.v., and an "abrupt" drop in acuity for all higher tones; and *2.* those with a "gradual" decline in thresholds, beginning with the low tones, for the successively higher tones. These two types of audiograms are similar to those of Control Group II, the only difference being in the hearing acuity for low tones. The remaining six ears had about the same degree of impairment of hearing for all tones; their audiograms are best described as "horizontal."

Seven, or one-fifth, of the ears have partial atrophy of the nerve in all turns of the cochlea, less severe in the apical than in the basal turn. For these seven ears there is good agreement, along the lines of the classical theories of hearing, between the cochlear lesions found and the hearing tests. Six of the seven have audiograms of the "gradual" decline type. The audiogram of the other ear (No. 35), in which only the apical third of the organ of Corti is normal, shows an extreme degree of "abrupt" drop in hearing for tones above 256 d.v.; the highest frequency heard at all was 1,024 d.v.

Seven other ears have middle-ear or otosclerotic lesions more severe than have been found in any ears with good hearing for all tones. Even though the Rinne test was positive, the impaired hearing for low tones must have been caused by the conductive lesions. Five of the seven ears have cochlear lesions in the basal turn sufficient to explain the impaired hearing for high tones. The other two had audiograms of the "horizontal" type.

Five ears are grouped together because each has a middle-ear lesion about as severe as has been found in any ears with good hearing for all tones and each also has a cochlear lesion

of the basal turn that, by itself, would explain only the impaired hearing for high tones. The observations strongly suggest that the combined effect of such middle-ear and basal-turn lesions is to cause an impairment of the hearing for low tones that would not result from either lesion separately.

Comparison of the cochlear lesions of the ears of Control Group II (impaired hearing for high tones only) with those of the present material also suggests that possibly the upper basal turn participates in the hearing of low tones.

In only four of the 35 ears is there no lesion to explain the impaired hearing for either the high or the low tones. For these cases, as well as for the others, the limitations of the histologic technique used must be kept in mind, also the fact that it has not been possible to study the central nervous systems.

Thirteen of the 14 ears with audiograms of the "abrupt" drop type have areas of atrophy of the organ of Corti in the basal turn. Partial nerve atrophy is the striking lesion in the group of 15 ears with a "gradual" decrease of hearing for the successively higher tones. These characteristic lesions are the same as were found by Crowe, Guild and Polvogt in ears with the respective types of audiograms and impairment for high tones only. The prominent lesions in the six ears with "horizontal" audiograms are of the middle ear.

The evidence from the present material as to tonal localization along the basilar membrane for the frequencies of 8,192, 4,096 and 2,048 d.v. is in complete agreement with the conclusions of Crowe, Guild and Polvogt. The area of most importance for 8,192 d.v. is in the middle of the lower basal turn; for 4,096 d.v., in the upper end of the lower basal turn, and for 2,048 d.v., in the lower part of the upper basal turn. The evidence as to localization for lower tones, on the contrary, does not support the theoretical views that there is even a moderately restricted area of response to these frequencies.

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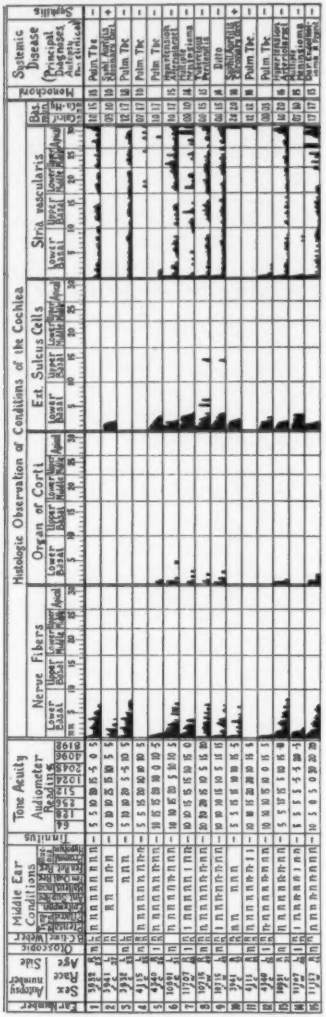
Johns Hopkins Hospital.

Note: For charts and explanations see following pages.

Chart 1. Data for the 15 ears of Control Group I.



EXPLANATIONS OF CODES AND ABBREVIATIONS USED IN CHARTS 1-2.

The acuity of hearing for the octave tones with frequencies from 64 to 8,192 d.v. is recorded in terms of the readings of a Western Electric Co. audiometer or as **nh** (meaning that the tone was not heard at the maximum intensity produced by the instrument used). Those ears with five-digit numbers (under the column heading of Autopsy Number) are from the pathology service of the Johns Hopkins Hospital and were tested with a 1-A audiometer; those ears with four-digit numbers are from the pathology service of the Bay View Division of the Baltimore City Hospitals and were tested with a 2-A audiometer.

Atrophy of the cochlear structures listed is shown in black. Complete filling of the space for any structure at any position on the chart means that the serial sections show total atrophy of the structure at the corresponding level. For the nerve, the external sulcus cells and the stria vascularis, the degree of partial atrophy is shown by the proportion of black to white at the corresponding position on the chart. For the organ of Corti the code is somewhat different. The condition termed "almost total" atrophy by Crowe, et al. (a hillock of undifferentiated cells on the basilar membrane) is charted by black across three-fourths of the width of the space. Black across half the width of the space means that there are no hair cells in the region, but that the general shape of the end-organ is still retained; and filling of one-fourth the space with black means that part of the hair cells in the region are missing. The latter is the slightest degree of atrophy of the organ of Corti that can be definitely differentiated in this material from artefacts of preparation.

For recording conditions observed in the several parts of the middle ear the following code is used: **n** means that the region is histologically normal; **n-**, **1**, **2** and **3** are used for lesions, according to severity. Except for **3**, these lesions are within the limits of those sometimes seen in ears with good hearing. Descriptions of many of the lesions are given in the text for the respective ears. The same code is used to record the appearance of the tympanic membrane at the time of otoscopic examination.

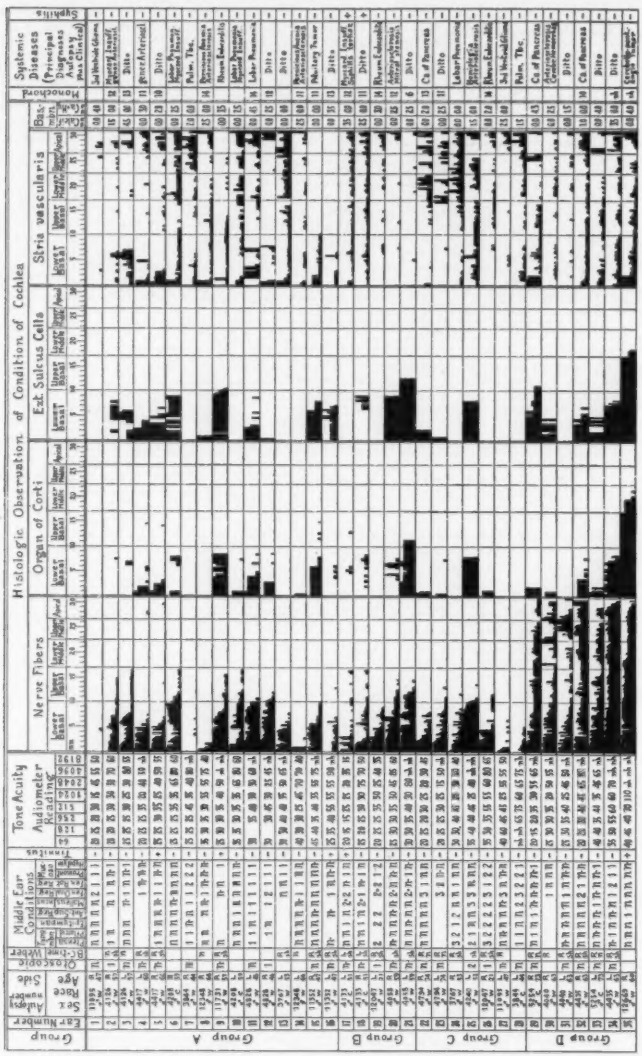
Under the heading of basilar membrane (Bas. Mbn.), the first column gives the length, in mm. from the basal end, of the area with the appearance of calcification (Calcif.), and the second column the total length of the area with either calcification or hyalinization (Ca. + Hy.) changes.

The figures in the column headed monochord are the upper limit of hearing by air conduction, as measured with a Struycken monochord, in terms of thousands of double vibrations per second.

Race is recorded thus: W—white; C—colored (American negro).

The other information in the charts is recorded by self-explanatory abbreviations or symbols.

Chart 2. Data for 35 ears with impaired hearing for all tones and with the Rinne test positive.



MENINGITIS (OTITIC) DUE TO TYPE III PNEUMOCOCCUS OPERATION, SULFANILAMIDE AND INTRASPINAL INJECTION OF PATIENT'S OWN SERUM; RECOVERY.*

DR. SAMUEL W. GARFIN, Boston.

The type III pneumococcus has long been recognized as a distinct entity in otology (Neuman-Ruttin[1]), in that it produces a more or less distinctive type of otitis media and mastoiditis.

Clinically, the onset of "mucosus otitis" is characterized by painlessness and absence of fever. There is some impairment in hearing, tinnitus and sensation of fulness in the ear. The membrana tympani may show only insignificant changes, such as slight opacity with reddening along the malleus, not unlike a mucous catarrh. The term "porky drum" quite answers the description. Frequently, no perforation occurs and the condition apparently subsides, though deafness, slight headache and a sensation of fulness persist. After a latent period, varying from several weeks to three or four months, symptoms of mastoiditis or intracranial complications appear. Since the infection has the tendency to burrow deeply towards the intracranial structures and away from the squama, there is rarely any mastoid tenderness. The discharge, if any, is usually scanty, sticky and mucoid in character.

The organism has characteristic bacteriologic, immunologic and epidemiologic features. Because of its large capsule and its tendency to grow in chains and produce a large waxy or mucoid colony on the surface of solid mediums (blood agar[2]), it has been known variously as *"Streptococcus mucosus capsulatus,"*[2a] *"Streptococcus capsulatus,"*[3, 3a] *"Streptococcus mucosus"*[4] and *"Pneumococcus mucosus."*[4a] This organism is now recognized as the type III pneumococcus. It has the same morphological and cultural characteristics as other pneumococci.[5]

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*From the Aural Service, Thorndike Memorial Laboratory, and the Neurological Unit of the Boston City Hospital.

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Type III is the most common type of pneumococcus found in persons in the normal nasopharynx and in patients with chronic infections of the respiratory tract and middle ear (Gundel and Linden[6]). Among 75 cases of otitis media and/or mastoiditis due to specific types of pneumococci, 37, or about one-half, were due to type III (Finland[7]). Wirth[8] found the type III pneumococcus responsible for 23.8 per cent of cases of acute middle-ear inflammation.

In 99 cases of pneumococcic meningitis occurring at the Boston City Hospital between November, 1929, and June, 1936, 25 were of otitic origin, 15 of which were due to type III. None of these cases survived (Finland[7]).

In the literature, there have been reported five cases of type III pneumococcus meningitis that have been treated by various methods and recovered. Only three of these were of otitic origin.

Allman's[9] case was treated by a radical mastoidectomy and labyrinthectomy. Steinholz and Gleich[10] used antipneumococcus serum intraspinally and intracisternally. Cavenagh[11] performed a radical mastoidectomy, administered 15 cc. of antistreptococcus serum intrathecally before the culture showed type III. After this, he used antipneumococcus serum (type not stated) intrathecally; recoverey occurred in five weeks. This patient also had lobar pneumonia. Weinberg[12] reported the recovery of a patient treated with retention enemas of potassium permanganate as advocated by Nott.[13] This was not an otitic meningitis. Ratnoff and Litvak[14] report the case of a girl, age 7½ years, treated for the first five days with antipneumococcus serum intraspinally without result. This was followed by intraspinal injection of ethyl hydrocupreine hydrochloride in addition. There was immediate improvement, which continued for three days, at end of which time the patient complained of blurred vision; the injections were now discontinued, but daily lumbar punctures were carried out with injections of 20 cc. of antipneumococcus serum. The authors believe that ethyl hydrocupreine hydrochloride enhances the curative value of antipneumococcus serum. This was not an otitic meningitis.

Bratesco and Moga[15] report a cure of pneumococcic meningitis (type not mentioned) by mastoid operation and daily injections of 2 gm. of utropine intravenously. From the clini-

cal description, it is hardly likely that this was a type III pneumococcus.

Kopetzky[16] mentions a cure of type III pneumococcic meningitis treated by: operation, repeated small whole-blood transfusions, and the administration of sulfanilamide. This case is soon to be reported.

The present paper concerns a boy, age 10 years, who recovered from an otitic meningitis due to type III pneumococcus. This is one of the six cases of cured pneumococcic meningitis reported by Finland and his co-workers[23] and the only otitic meningitis of this type III on record at the Boston City Hospital to recover.

The method of treatment employed here is based upon the results of clinical bacteriological and immunological studies in patients with pneumonia and other pneumococcic infections, including meningitis, carried out by Finland and his co-workers. Also included in this study is the more recent investigations of the mechanism of the action of sulfanilamide alone or in combination with specific serum on pneumococci in human blood *in vitro* and in patients undergoing treatment for pneumococcic infections.

Sufficient experimental[17, 18, 19, 20] and clinical[21, 22, 23] evidence has now been accumulated to show that sulfanilamide or its derivatives administered, in appropriate amounts, exercises an inhibitory (bacteriostatic) effect on most of the strains of pneumococci tested. The bacteriostatic action may fail or is only slight in the blood of certain patients with pneumococcic infections, particularly if the blood stream is heavily invaded.

From the studies of Finland and his co-workers,[23] it has been shown that, following the administration of sulfanilamide, a decrease of the meningeal infection takes place, at least temporarily. Some cases may recover with this treatment alone, but most cases require the presence of antibody and complement; however, complement is not found in the spinal fluid, and after (horse or rabbit) antiserums are given intravenously, even in the presence of only small numbers of pneumococci, the antibody does not reach the cerebrospinal fluid in demonstrable amounts. For this reason, it is apparently necessary, in most instances, to introduce some antibody into the infected area.

After considerable cerebrospinal fluid is drained off and the infection reduced by the use of sulfanilamide, "very small amounts of specific antibody should be sufficient to complete sterilization of the cerebrospinal fluid, provided that there have not yet developed any well localized areas of abscess formation."

The most readily available supply of antibody, and one least likely to produce unfavorable reactions, and at the same time provide complement and an optimum amount of antibody, is the patient's own fresh serum, after a balance of sufficient antibody has been established in the circulating blood.

The following is the general procedure adopted by Finland and his co-workers[23] for the treatment of pneumococcic meningitis and carried out at our hospital:

1. Complete and frequent drainage of the spinal fluid.

2. Immediate institution and continuous use of large doses of sulfanilamide by mouth, or by subcutaneous injection if necessary. The optimum dose has not been determined.

3. Identification of the pneumococcus as rapidly as possible and the intravenous administration of sufficient specific antipneumococcic serum to establish a balance of antibody in the circulating blood.

4. Moderate fluid intake by whatever route necessary to insure adequate amounts of spinal fluid for drainage.

5. About two hours after a reasonable dose has been given, blood is withdrawn from the patient and serum rapidly separated.

6. Following the removal of spinal fluid at the time of the next lumbar puncture, this serum is given intraspinally (5 to 10 cc. should be sufficient. The optimum amount has not been determined but it is probably small). This procedure may be repeated with subsequent lumbar punctures if necessary. In small children or in infants, it may be necessary to use fresh human serum from another source for complement and, in that event, only very small amounts of therapeutic serum need be added; 0.5 cc. or less should be sufficient.

7. Lumbar punctures are repeated until the fluid is normal. The frequency is determined by the initial pressure of the fluid and its cellular and protein contents.

8. Transfusions are given after the first week of sulfanilamide therapy if anemia develops and are repeated, as necessary, until the drug is discontinued (after seven to 14 days of sterile fluids).

These (Finland) procedures serve to insure a balance of antibody in the blood stream and control of bacteremia. They should, in most instances, where the sulfanilamide effectively reduces the infection, provide an adequate amount of antibody and complement in the optimum proportions and in a medium which is likely to give the least local or general reactions and the greatest antibacterial effect. The case to be presented will illustrate the application of some of the principles enumerated. It also illustrates the spontaneous development of antibodies in a patient with infection while under sulfanilamide treatment. This phenomenon made the administration of therapeutic serum unnecessary, the patient's own serum having sufficient antibodies for the purpose.

CASE RESUMÉ.

A white school boy, age 10 years, was referred by his physician, Dr. E. E. Bowen, and admitted to the hospital on the night of April 9, 1938, complaining of frontal headache and fever of three days' duration. On the day of admission, he began to vomit and developed a stiff neck. One year previously, he had acute otitis media and since that time had purulent discharge off and on from the left ear. There was some dried purulent exudate in the external canal; the drum was practically totally destroyed except for Shrapnell's membrane. There was no mastoid tenderness. Culture of the exudate from the left ear showed type III pneumococci. There was some hyperemia of the nasal mucous membranes. The neck was rigid and the Kernig and Brudzinski signs were positive on both sides. Roentgenograms showed the mastoid tips to be clear. Lumbar punctures done on admission and again on the following morning showed ground glass fluid under pressure of 300 and 370 mm. of water with 3,000 and 5,100 cells per cmm., respectively, mostly of the polymorphonuclear variety. Cultures of these fluids showed no growth and smears of the sediment showed no organism. Sulfanilamide was given by mouth beginning the morning of admission — 20 gr. every three hours, approximately 6 gr. during this day (see Fig. 1).

At this time, the morning of April 10, the patient was seen by a neurological consultant and by Dr. L. M. Freedman, Chief of the Aural Service. Both consultants advised immediate operation. A modified radical mastoidectomy was performed on April 10. Considerable necrotic tissue was encoun-

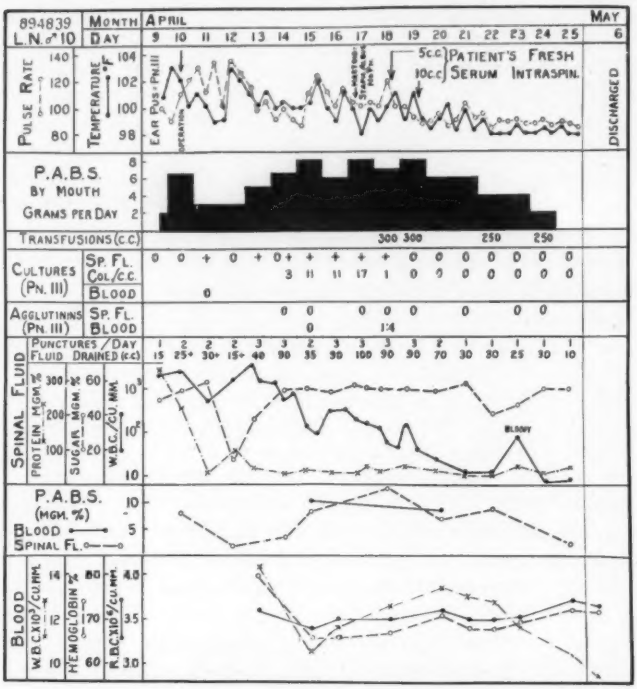

Fig. 1. Temperature, pulse rate, essential therapy and laboratory findings. Abbreviations and Symbols: P.A.B.S.=sulfanilamide (para-amino benzene sulfonamide). Cultures PN.=pneumococcus; the type is indicated in Roman numerals. SP. FL.=spinal fluid. Col. /cc.—number of colonies per cc. in blood agar pour plates=growth in broth of the type of pneumococcus indicated.

tered in and around the antrum, which was situated high and close to the tegmen. Mucoid, stringy pus under pressure appeared from the antrum. The cells of the perilabyrinth and those along the facial canal were also necrotic and were removed. The dura of the middle and posterior fossa, including the lateral sinus, was well exposed. The posterior canal

wall was partially removed and the tegmen tympani was likewise removed.

Lumbar puncture was done after the operation was completed. A culture taken from the antrum at the operation showed pneumococci type III.

On the day following operation the patient appeared considerably improved. There was a drop in temperature, the headache, stiff neck and vomiting disappeared.

Lumbar puncture was carried out twice daily and the administration of sulfanilamide was continued (see Fig. 1). Except for the first two spinal fluids obtained on the first and second day of admission, the lumbar puncture from April 11 to April 18 yielded fluid from which type III pneumococci were cultured.

On April 14, the patient was transferred to the neurologic service and intensive sulfanilamide therapy was begun. The neurological service made the following comment at the time of transfer: "Physical examination and neurological examination were precisely the same as that on the aural service, except for the mastoid, which was draining pus at the time of the transfer, April 14. Patient appeared to be in excellent health condition, so much so that in spite of positive culture obtained on the eleventh and thirteenth, the diagnosis of pneumococci meningitis seemed unlikely."

Diagnosis: Pneumococcic meningitis III.

POSTOPERATIVE MASTOIDECTOMY.

The blood serum of the patient on April 15 failed to show agglutinins for type III pneumococci but they were demonstrated in the serum of April 18. On the latter date, the culture of the spinal fluid was still positive for type III pneumococci, and after this fluid was withdrawn, 5 cc. of the patient's own freshly prepared serum was injected intraspinally. All subsequent lumbar punctures yielded fluids which showed no growth on culture. A second intraspinal injection of the patient's fresh serum was given on April 19; thereafter, the patient made an uneventful recovery. Mouse protection tests carried out later showed that the serums of April 15 and April 18 in 0.2 cc. amounts protected mice against 1,000 and

10,000 fatal doses of type III pneumococci, respectively. The spinal fluids of the same dates showed no protection.

SUMMARY.

1. A case of type III pneumococcus otitis, mastoiditis and meningitis is described.

2. Marked clinical improvement followed mastoidectomy and intensive sulfanilamide therapy, but failed to bring about sterilization of the spinal fluid.

3. Complete and permanent cure of the meningeal infection occurred after the first intraspinal injection of patient's own fresh serum, which contained antibodies actively developed by the patient.

The author acknowledges his indebtedness to Dr. Maxwell Finland and his co-workers, who directed and carried out the details of the treatment and carried out the bacteriological and immunological studies in this case in the neurological unit and at the Thorndike Memorial Laboratory of the Boston City Hospital.

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485 Commonwealth Avenue.

ACUTE ANTERIOR POLIOMYELITIS WITH SPECIAL REFERENCE TO ITS RHINOLOGICAL ASPECT (LANTERN SLIDES DEMONSTRATION).*

DR. LEON FELDERMAN, Philadelphia.

Of all the infectious diseases to which mankind is subject, acute anterior poliomyelitis takes first place for its suddenness of attack, the helplessness of the patient and the terror it strikes into the hearts of parents. A child, apparently in normal health, within a few hours is seized with characteristic symptoms.

Hyperesthesia, fever, headache, vomiting, costiveness more often than diarrhea, drowsiness and irritability when disturbed, impaired power of locomotion and meningeal symptoms, are but a few symptoms of this much feared malady.

The course of poliomyelitis may be divided into three stages: *1.* A generalized infection with systemic involvement; *2.* symptoms which involve the central nervous system, particularly the meninges; and *3.* impairment of the nervous centre of the peripheral motor neurons. These stages are not sharply defined in every instance and frequently overlap one another.

It appears that each year epidemics of this malady are becoming more widespread, leaving as an aftermath crippled limbs in despairing numbers. Physicians, as well as the lay public, are gravely concerned over these recurrent epidemics, and are particularly interested in the study of their prevention.

Acute poliomyelitis is rarely seen in the Spring, but attains epidemic proportions during the Summer months and in the early Fall. In the North, where temperature instability is sharper, the epidemic is regularly more severe than in the South. Interestingly enough, the inception of the epidemic is associated with meteorological changes. It is also to be noted

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that it is more marked among those individuals who suffer from head colds and sinus infections.

Most epidemics, such as that of 1916-31, have had their beginnings along the seaboard cities. The crowded beach resorts, with their numerous swimmers, make infection easy and isolation difficult.

In 100 cases studied, Sotos and Rubin[1] point to the peak of hospital admissions as occurring during the last week of August and the first week of September, which fact seems to support the observations of Aycock.[2]

The ages of the patients vary from six months to 12 years, with the average age of four years. The male, with a percentage of 55, has a slight lead. The negro, judging from available statistics on the subject, is slightly less susceptible than the white man. In the 1931 New York City epidemic, the attack rate among negroes was only two-fifths of that of the white population. Out of 2,402 cases admitted to the contagious disease hospitals, only 74 were negroes (Smith and Landon[3]).

Aycock and Luther[4] mention the onset of poliomyelitis in 16 instances following tonsillectomies and adenectomies; Silverman,[5] in 1931, reported five such cases, and Fischer and Stillerman,[6] 1935, in a group study of 686 cases, reported 10 cases within a period of four weeks following operation.

Recent experimental studies of poliomyelitis show that the virus is neurotropic, particularly for the olfactory bulb. Once this area is infected, the virus spreads along the axonal routes. In support of this, experiments with the Macacus rhesus create interesting studies, in which typical symptoms and lesions of poliomyelitis are developed.

A radical step in animal experimentation was undertaken by Jungeblut and Spring,[7] in which the spinal cord was transected at the level of the first lumbar vertebrae prior to the introduction of the virus, and nine days after the inoculation the monkey developed typical lesions of cervical and thoracic cord only, the spinal cord which was previously sectioned remaining free from the infection.

Faber and Gebhardt[8] have been able to recover the virus from the olfactory bulb during the incubation period, follow-

ing the infection of the intranasal area, and have corroborated this theory of axonal dissemination. As a result of these experiments, the matter of the propagation of the virus through the olfactory bulb is no longer a debatable question.

Harbitz-Scheel-Muller-Wickman[9] found that the preponderance of lesions is in the central ganglia, midbrain and medulla, which seems definitely to point to the fact that the central nervous system carries the brunt of infection.

Flexner and Clark[10] found that, following the inoculation of the virus through the olfactory lobe, the monkeys developed the infection within 24 hours.

To further prove the theory that the virus makes the journey along the olfactory nerves, they took another series of monkeys and, after partially removing the bulb and tract, made repeated virus inoculations, but with no success, whereas the control animals succumbed very often to a single inoculation. This establishes the thought that the infection is neurotropic and that the virus present along the olfactory nerves is carried to the central nervous system, and propagated along the nerve tract with its attendant syndrome.

Sabin and Olitzsky[11] have conducted experiments on 18 monkeys by instilling 1 cc. of 10 per cent virus suspension into each nostril and repeating the performance 48 hours later. Between the fourth and sixth days, pronounced symptoms of paralysis resulted. These investigators made pathologic examinations as early as the fourth day, and discovered changes in the olfactory bulb diencephalon, mesencephalon and pons, the spinal cord remaining quite normal. Once the paralysis set in, the changes were more extensive, of course, varying with each monkey. The virus of poliomyelitis produces characteristic lesions in the olfactory bulbs of rhesus monkeys when it invades the central nervous system by way of the olfactory nerves.

The experiments of Lennette and Hudson,[12] where they sectioned the olfactory tracts of five monkeys and then intravenously inoculated them with 1 cc. of 10 per cent poliomyelitis virus on each of three successive days and the monkeys remained well, and of the five control monkeys similarly treated, four immediately succumbed to poliomyelitis, is indeed a classic.

When the acid phosphate solution was not used as a nasal wash, 16 out of 24 monkeys succumbed to poliomyelitis. When the acid phosphate was used, all of the nine monkeys succumbed to a lesser quantity of virus injection.[13]

Schultz and Gebhardt[14] report successful "takes" in 43 out of 46 monkeys inoculated through this olfactory pathway. As early as the fourth day of virus invasion, they were able to obtain a potent poison only from the olfactory tracts, other parts of the central nervous system remaining free from the infection.

Toomey[15] directs attention to the probable pathways of the VIIth nerve for the transmission of the virus of anterior poliomyelitis; however, this is not by any means conclusive.

Fairbrother and Hurst[16] established the fact that, throughout its entire course, poliomyelitis is primarily and essentially an infectious disease of the central nervous system, caused by a strictly neurotropic virus.

Wilder[17] substantiated the neurophilic theory by showing that the virus invades the olfactory epithelium and gradually works its way along the axons of the nervous system to the medulla and spinal cord, consuming about seven days in its labyrinthian course.

Armstrong and Harrison[18] found that nine out of 10 monkeys, where picric acid was instilled intranasally, could be protected against intranasal inoculations of the virus which produced poliomyelitis in nine out of 10 control cases. Their conclusion was that picric acid has protective power against poliomyelitis.

The work of Fairbrother and Hurst, Yungeblut and Spring, and Faber and Gebhardt demonstrates that the pathogenesis of poliomyelitis is a neurotropic virus of the intracellular type which has the "inside track" of the neurons and makes its point of entrance and exit at will.

The conclusions derived from such experiments suggest strongly that the usual journey of the infection of poliomyelitis is by way of the olfactory tract.

Landsteiner and Levaditi[19] showed that tonsils may harbor the virus of poliomyelitis, and more recently Aycock and Luther[19a] have reported a relationship between tonsillectomies and the onset of poliomyelitis.

It was reported several years ago that the virus gains entrance in the pharyngeal region and, becoming localized there, secretes a toxin possessing neurotropic properties like the tetanus or diphtheria germ.

The droplet theory of virus transmission is gaining currency as borne out by experiments performed in monkeys. It is a fact that the virus' greatest concentration point is in the nasopharynx in subjects infected with the disease. The experimenters stress the theory that the virus which finds access to the central nervous system and to the spinal cord does so by invading the nasopharynx and by direct extension of the olfactory nerve fibres.

From the experimental, epidemiologic and clinical viewpoint, the nasopharynx constitutes the portal of entry for the poliomyelitis virus.

In view of the widespread interest existing today in the outcome of chemoprophylaxis of the olfactory bulb region, the following quotation seems pertinent:

"This organ is situated at the upper part of the nasal cavities. Here, the mucous membrane covering the superior and middle turbinate bones and the corresponding part of the septum is different from that covering the remainder of the nasal passages. In the lower part of the nasal cavities the mucous membrane is of the ordinary respiratory type and is composed of ciliated columnar epithelium, containing a number of goblet cells. In the olfactory region the epithelium is much thicker, yellowish in color and, apparently, composed of a layer of columnar cells resting on several layers of nuclei. In human beings the olfactory organs are located in each nasal cavity. These cavities are separated by a nasal septum, which acts as a partition between the two nasal fossae. As stated before, they have an external opening, called the anterior naris, and an internal opening, the posterior naris or choana. The lateral wall of these cavities are a series of folds, called nasal conchae. They are usually three in number: The inferior, the middle and the superior; sometimes one may find a fourth concha, called the supreme concha. According to Schaeffer, this concha may be observed in about 60 per cent of all human beings.

"The olfactory nerve is composed of approximately 20 nonmedullated filaments, which pass through the foramina of

the lamina cribrosa, of the ethmoid bones into the anterior cerebral fossa and, after piercing the meninges, make up the olfactory bulb and become intracerebral. These fine filaments penetrate into the brain covering the olfactory bulb, which in turn synapse with the dendrites of the olfactory pathway. The final portion of the olfactory nerve becomes the terminal nerve. The latter has been demonstrated in the cadavers of infants and adults. The peripheral twigs can be found along the ethmoid bone, where the nasal mucous membrane joins the olfactory regions. The central organ of the 'smell brain' (rhinencephalon) in point of evolution, phylogenetically, may be regarded as the oldest brain. Physiologists refer to it as archi pallium.

"The olfactory cerebral cortex falls into two groups, namely: *a.* Olfactory cerebral cortex; *b.* olfactory reflex centres. The olfactory bulb in man exists in a rudimentary state. It is elongated and oval shaped, yellowish in color; is situated in the lamina cribrosa of the ethmoid bone and gives origin to olfactory tract 18 to 20 mm. long. The olfactory bulb and tract lie close by in relation with the roof of the nasal cavity. The bulb rests upon the cranial surface of the lamina cribrosa of the ethmoid bone. Not infrequently it is encroached upon by the irregularity of the frontal sinus, such as the cells of the crista galli or ethmoid cell. This anomaly may help to explain the anosmic condition. We have three kinds of olfactory epithelium, *viz.*, *a.* basal cells; *b.* ordinary epithelium; *c.* sustentacular cells or sense cells.

"The olfactory receptors are, therefore, represented by a series of specialized end-organs, which receive impressions borne through air or water, or both. These end-organs are stimulated by aromatic and volatile substances, which act upon the specialized olfactory mucous membrane of the nose. The peripheral organ of smell consists of an epithelium containing sensory cells, which give origin to fibres forming the olfactory nerve. These cells are bipolar. Their central processes enter into the formation of the fila olfactoria, while their peripheral processes are equipped with a number of hair-like prolongations at their extremities, called the olfactory hairs."

The medical profession has been engaged in the task of prevention. At first, vaccines prepared for the virus were

introduced. The ricineolated and formalized viruses are today discarded as valueless. The vogue of vaccine therapy was, happily, short-lived, since it was possible to reproduce the disease in monkeys which had been previously serum-logged with one form of vaccine or another.

"Since serum cannot be depended on to block the implantation of virus, and since an infection once established cannot be modified by immune serum, it appears that the only remaining line of defense against this disease which is at all promising lies in the possibility of active immunization. It should be remembered, however, that for poliomyelitis the term 'active immunization' has a special significance which may be overlooked. The basic feature of an active immunization against poliomyelitis is modification of the susceptibility of neurons (tissue immunity). There is an important difference between a tissue immunity, in which antibodies are an incidental by-product of infection, and an 'immunity' in which antibodies represent the sum of the defense afforded."[21]

Disappointing as were the failures of an immune serum, there arose the new method of dealing specifically with the olfactory bulb. Although the editorial policy of the *Journal of the American Medical Association* was by no means enthusiastic, it reserved final judgment, pending a fair trial of this method.

"The successful use of a zinc sulphate spray in the prevention of experimental poliomyelitis in monkeys by Armstrong, Sabin, Schultz and their co-workers has stimulated much interest in its effectiveness as a preventive of the disease in human beings. Schultz demonstrated at Stanford University that, in the experimental infection a solution containing 1 per cent of zinc sulphate, 1 per cent of pontocaine and 0.5 per cent of sodium chloride in distilled water is most effective in the monkey. The use of this technique in the human being has been too variable and uncontrolled to permit even an approximate estimate of its value. . . . If the evidence obtained this year is in any way encouraging, plans for determining the effectiveness of the attempted preventive might be worked out so that something resembling a serious scientific experiment may be tried next June, July and August."[22]

In dealing with preventive agents, one must not wax enthusiastic. Medication which can be applied to monkeys with

impunity may not be practicable in man. Apropos of this thought, it is interesting to quote a letter from one of our confrerés, who was one of the many to recognize the impracticability of the zinc sulphate spray:

"The excuse for this letter is the difficulty which the deleterious effects of an exploited remedy have in catching up with its recorded virtues.

"My first opportunity to employ zinc sulphate for the prevention of poliomyelitis was for the protection of the mother of a little patient. The instructions with regard to material, instruments and technique, as recommended in the Peet article, were carefully carried out. The mucosa was shrunken with epinephrine and the zinc sulphate 1 per cent, sodium chloride 0.5 per cent, pontocaine hydrochloride 1 per cent solution was applied under visual control to the olfactory area. The young woman commented that she felt as if she were going to have her tonsils out again, but only after leaving did she begin to be distressed. She complained that the sensation in her nose was like red hot irons. It lasted through the night, the application having been made early in the morning, and the next day she still had a marked rhinorrhea. She refused a further application. I did not use 2 cc.; to me, it seems that 1 cc. per nostril is an enormous amount to apply with an atomizer, even in an adult nose.

"If the treatment proves satisfactory and continues to be recommended broadly to the medical community, it seems to me that advice as to how to modify these unpleasant effects should go with the recommendation for the treatment. I have had one case, and that is all I am going to have until or unless the efficacy of the treatment and its essential harmlessness are more firmly established.

"I hope that the unpleasant effects may be modified or eliminated."[23]

The following quotation clearly condemns this method of spraying the olfactory bulb:

"The serious outbreak of poliomyelitis in and around Toronto this year afforded an exceptional opportunity for the study of the prophylactic value of zinc sulphate sprays. The report by Tisdall and his co-workers on this subject deserves careful study. A trial of nasal spraying was approved by the Depart-

ment of Health of Ontario on Aug. 29. It was agreed that each child should be sprayed on two occasions, 10 or 12 days apart, and that from 0.5 to 1 cc. of the solution should be placed in each naris in each spraying. The solution contained 1 per cent zinc sulphate, 1 per cent pontocaine and 0.5 per cent sodium chloride. The spraying was done according to the technique of Peet, Echols and Richter, but differed from their recommended procedure, in that it was not administered on three successive days, since this method was considered impracticable. The peak of the epidemic occurred during the week ending Sept. 4. The first spraying, begun on Aug. 31 and completed by Sept. 5, was performed by 55 attending otolaryngologists of eight hospitals in the city. In the period of one week from the authorization of the study, 5,233 children had received the first spraying. The second spraying extended from Sept. 13 to Sept. 16. No serious complications occurred.

"Nine hundred and nine tests, representing 750 children, were made to determine the number who had lost their sense of smell. From these observationse it would appear that some 25 per cent had anosmia. A representative control group of 6,300 children was obtained in the city proper. Among the 4,713 children who were sprayed twice, 11 cases of poliomyelitis occurred up to Oct. 12, 30 days from the second spraying. One of these occurred six days after the spraying and was not included in the analysis. In the control group of 6,300 children, 18 cases occurred. The attack rate in the period from seven days after the first spraying to 30 days after the second spraying was 2.1 in the sprayed group, and 2.9 in the control group. The difference is not statistically significant. In the total of the city and suburban groups, the attack rates were 2.9 in the control group, and 1.8 in the sprayed group, which also was found not to be statistically significant. The report concludes that, since the spraying method employed in this study must be conducted by otolaryngologists or other physicians especially trained in intranasal treatment, requires special facilities and cannot be done quickly enough to meet the emergency of an outbreak, it cannot be considered a practical public health procedure."[24]

Notwithstanding the fact that physicians in this country are wholly in agreement with the opinion just cited, investigators continue, by trial and error method, to experiment on mice with chemical agents such as cobra venom, sodium chlo-

ride, distilled water, alum formalin, glucose, ferric chloride, aluminum chloride, picric acid, tannic acid, lead acetate, sea water, thymol, tribromphenol, picramic acid, dinitrocresol, dinitrophenol and quinine hydrochloride, and other chemicals.

Rake made exhaustive studies with tannic acid and arrived at this conclusion:

"That prussian blue particles pass rapidly from the surface of the olfactory mucosa and within two minutues are found in the tissue spaces, in blood and lymph vessels, in the perineural spaces of the olfactory nerve tissues and in the subarachnoid space and pia-arachnoid membrane. Preliminary treatment of the olfactory mucosa with tannic acid does not alter the speed with which this absorption occurs; however, it does prevent an inflammation from entering the olfactory mucosa and reaching the tissue spaces, the vessels and the subarachnoid space with the same rapidity as the pigment. They evade by passage between the cells of the mucosa and there is no apparent affinity of the organism for the olfactory sensory cells. Tannic acid treatment of the olfactory mucosa in no way alters this invasion of organism through the mucosa."

Rake[25] is not firmly convinced that the tannic acid treatment of the olfactory nasal mucus is effective in blocking the portal of entry of the virus of poliomyelitis. It seems that the tannic acid treatment, as a prophylactic measure in this infection, can only hope to bring the following characteristic changes in the tissue: *a.* of color; *b.* of size (pathological hypertrophy); and *c.* of function. This treatment superimposes a severe chemical reaction on a pre-existing latent sinusitis and leaves the door open to further breakdown of these structures.

All life units are endowed with certain characteristics in their immediate environment. Every change in the environment registers in the living cell. When scientists speak of pathology, they must think of the physiological behavior of the organismal unit cell and what it must do to maintain its own physiological level. After all, the organism is just as strong as its surroundings will permit. The environment enmeshes the constituent cell of the individual with many stimuli of the normal and of the abnormal kind. To maintain a biological balance, the human cell must display three indis-

pensable characteristics: *a.* Irritability; *b.* respiratory action for gaseous exchange; and *c.* ability to synthesize.

Prof. Edward Lodholz[26] in his lectures speaks of the stimulus response interaction which is blended with the well-being of the organism. A chief characteristic of living matter is its ability to react to environment, as well as its capacity to incorporate certain selective materials and energy from the environment and transform it into living substances in a manner characteristic of that particular species.

The question has been asked as to the special features in the composition or constitution of living matter which render their chemical processes so susceptible to influence by changes in the surroundings. The answer is that living protoplasm consists of a large variety of chemical compounds held together by a complex architecture. Both its chemical composition and its structural or morphological constitution are to be regarded as chief factors in the determination of its special type of cell activity. The compounds in the living, therefore, proteins, carbohydrates, fats, salts and water, are substances which are oxidized to carbon dioxide and water.

It must be understood that the defense of the organism in the environment is just as proportionately strong as ability of the protoplasmic structure to resist injurious agents. The rate which affects the permeability and electrical polarization of the protoplasm is governed by its inner structure. Many forms of protoplasm are structurally unstable, causing cell disintegration when exposed to slight mechanical or other stimuli. Hence, physiology gives way to pathology in a fraction of a second.

Under normal conditions, first, the cell may be translucent, and then it takes on the capacity to gel, indicating coagulation, or changes in the cell matter. Finally, if this condition is not corrected, death of the cell ensues; and there also occur changes of physical consistency, loss of tensile strength, increase of viscosity, death and rigor or some of the associated factors which constantly beset the cells of the human organism.

One of the early signs of departure from the normal is diminished cell vitality, with a decrease of oxygen consumption. There is no doubt, however, that the high velocity of

the more characteristic chemical reactions of protoplasm (*e.g.*, the oxidation of sugars and amino acids) is chiefly determined, directly or indirectly, by the polyphasic structure of the living system. The structure of the human cell is a highly complex organism and depends for its growth, repair and maintenance upon the essentials already mentioned. Basic protoplasmic structure resembles an emulsion which contains two or more fluid phases of different composition, one (or more) of which are in a fine state of subdivision and suspended or otherwise dispersed in the other, which is continuous.

Physiological characteristics of these films are their semipermeability, the membrane becoming permeable to substances to which formerly they were, with difficulty, permeable or impermeable. All permeability increasing substances may be destructive to living protoplasm, and become cytologic agents. The protoplasmic film structure forms the chief controlling factor in the interchange of diffusible materials between the different regions of the protoplasm and between the protoplasm and the exterior. Once the cell permeability is disturbed, the colloidal equilibrium is unbalanced and that particular tissue degradates.

Surface conditions, too, represent controlling factors of primary importance in the chemical reaction of protoplasm. Response to stimuli may be simple or complex; either brings about a change of protoplasmic structure. Particularly are changes noted in nerve tissues and in cases of anterior poliomyelitis.

Some of the external factors include variation in temperature, oxygen supply, H-ion concentration, presence of specific chemical substances, mechanical conditions and light gravity.

In mammals, cell permeability may be due to changes of temperature, difference of osmotic pressure, difference of H-ion concentration, presence of special chemical substances, while relatively insensitive to other agents. This condition is illustrated by the various sensory receptors, such as smell, taste and vision. It is obvious that each cell organism has a special peculiarity of action and reaction, which depends upon the inherited organization of the cell structure. The response is both qualitative and quantitative. These phenomena appear to be indices or expressions of the fundamental physical and

chemical changes which occur in all irritable living forms during stimulation.

The ciliated epithelium is composed of cells which are the integral part of the upper respiratory system. Here, we are concerned chiefly with that portion of the respiratory organ which plays a rôle in this infectious disease. Much of the ciliated epithelium function has been clarified through the indefatigable work of Lucas[27] on the lower animals, particularly the monkeys (Macacus rhesus). He placed foreign particles within the noses of Macacus rhesus, which were subsequently propelled in oblique direction, downward and backward. These particles took this course, whether they were originally in the medial or lateral surface of the nose or in the nasopharynx. Bryant,[28] in 1916, and Yates,[29] in 1924, studied the sperm movements of 25 ciliary processes, and the former fixed this rate at 0.25 mm. per hour. Gravity, respiratory movement and position, as well as other factors, determine the movement and the function of the cilia.

In bacterial allergy, one can study the gamut of histopathological changes as they affect the ciliary processes.

"The cytologic structure of the upper air passages is composed, in the main, of ciliated epithelium, for the pseudostratified layer and the columnar layer, which line the nasal cavities and the paranasal sinuses, abound in ciliated cells. The vibratory area by means of an effective beat, governed as it is of to and fro movements, are uninterrupted, harmonious, with a polarity of the epithelial cells always in a constant position. The epithelial layer is covered by a relatively thin layer of mucus, which acts as a transport through the medium of the ciliary movement and thus secretions are propelled from the nasopharynx to the oropharynx and, finally, toward the esophagus and the gastrointestinal tract. This is one of the main avenues of transportation of the mucus which enmeshes numerous bacteria. Persistent chilling, bad aeration, chemical irritants, faulty metabolism greatly modifies the movements of the ciliated epithelium, recognized as highly specialized structure. The invasion of an organism results in a leukocytic exudation or an outpouring of free excretions of pus from the nose and sinuses, commonly diagnosed as the ordinary head cold. The constant and repeated injury to the fine and delicate mucous membrane and its adjacent struc-

tures can only result, at first in an inflammatory condition of the upper air passages when the normal physiologic mechanism is hampered."[30]

The parallel between the seasonal variation in the physiological function of the organism and the seasonal curve of poliomyelitis exists. This explains the fact that the human body is not able to cope with the stresses concomitant to seasonal variations. Then, too, the peak of poliomyelitis epidemic not infrequently corresponds to the pollen concentration in the atmosphere. The constant bombardment of the pollens on the nasal mucosa heightens the susceptibiiity of the allergics in the presence of such epidemics. That the endocrine dysfunction definitely plays an etiologic rôle in these infections was pointed out by Draper.

The key of prevention to poliomyelitis resides in the knowledge of the physiological behavior of the nasal ciliated epithelium. Any factor which interferes with ventilation and drainage of the accessory sinuses of the nose will imbalance the harmonious relations of these nasal "hair brushes" and predispose the patient to infection, whether from germ or virus. Proetz, by studying 50 specimens of the accessory sinuses, arrives at this conclusion:

"If, in the (probably unsuccessful) attempt to reach the infection in the submucosa, one paralyzes or destroys the overlying cilia, he has disabled his best ally. It is important, therefore, to select with some care the drugs to be used, especially in the presence of acute infection."[31]

The lay use of the popularly advertised oleaginous and non-oleaginous solutions, as a panacea for head colds in infants and children, makes the child an easy prey to much more dangerous infections than those for which the alleged cure is applied.

Swimming, diving and overheating of houses and apartments are but a few factors which rob the individual of the natural protective power.

Although strong chemicals may afford a limited protection to the patient, they actually divest him permanently of the highly specialized sense of smell. It is important that solutions applied to the nasal mucosa do not alter the normal Ph, and that there be maintained the function of the ciliary proc-

esses, which is the infantry in our first line of defense. Some of the solutions are inexpensive, painless and easy to apply. There are many solutions to be considered: neutral acriflavine, 1:8,000 strength, using normal saline as a base, has definite protective virtues.

A chemotherapeutic agent that will not slow up the ciliary movement is the choice in prevention of poliomyelitis. Finally, the problem of poliomyelitis is a rhinological problem and, as such, should be the study of those trained in this special field.

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Central Medical Building.

DISCUSSION.

DR. MOSES L. BREITENSTEIN: I find the theory of Dr. Felderman interesting, and I agree against the promiscuous use of nasal sprays in concentration that would interfere with the ciliary processes.

The following drugs or conditions affect the ciliary movements:

1. Heat, cold ephedrine, menthol, etc., agents which dry the mucous membranes slow down the ciliary movement.

2. Cocaine halts the ciliary beat.

3. Epinephrine halts it for good.

4. Drops in temperature to 7 to 12° C. and sudden changes upward cause the ciliary movement to stop.

5. Three per cent ephedrine-hydrochloride at times causes the movement of the ciliae to increase.

6. Five per cent cocaine does not impair the movement.

7. Silver protein is definitely bad.

8. One per cent menthol, thymol, eucalyptol are not good for the ciliae.

9. Silver nitrate is definitely fatal to them.

DR. HUNTINGTON WILLIAMS: The city of Washington decided against nasal sprays as a prevention to poliomyelitis, and I, too, share Dr. Felderman's views.

DR. HOWARD A. HOWE: I find Dr. Felderman's paper intensely stimulating, and I recommend further studies in this endeavor. I should like to ask Dr. Felderman, since he ties up poliomyelitis with allergy, how he accounts for the cases found in the far North.

DR. FELDERMAN, concluding: I wish to thank all those who participated in this discussion. With reference to Dr. Howe's question, I would like to remind him that when studying the numerous causes of allergy, we find that patients may be susceptible to inhalants (pollen), or ingestants (foods); then, too, the modern methods of transportation, such as automobiles and airplanes, account for the ragweed being carried to northern climes.

FOREIGN BODY IN NOSE (REPORT OF CASE).

DR. MORTIMER M. KOPP and DR. HAROLD LEVY,
Brooklyn.

The presence of foreign bodies in the nose has been quite frequently reported; however, it is unusual for a foreign body to remain unrecognized for a long period of time. Especially is this true in the nose, where examination is fairly easy. The present case is reported because the foreign body was in such a position that it could not be seen and was not suspected, despite frequent examination and treatment over a period of three years by a number of rhinologists.

The diagnosis was made when X-rays were taken to determine the condition of the nasal accessory sinuses.

Case Report: The patient, a girl, age 6 years, was first seen on April 30, 1938. The following history was obtained: The family history and that of the child's first three years of life were essentially irrelevant to the present story. At that time the child began to suffer with repeated colds and a "chronic running nose." She was not severely ill, however, until one year later, in the Spring of 1936, when she developed bilateral purulent otitis media following one of her colds. When this attack subsided, the tonsils and adenoids were removed. The child did well for a few weeks, but from then on there was recurrence and insidious progression of the rhinitis and the attacks of upper respiratory infection. Repeated and varied treatments were given locally to the nasal mucous membrane, with little or no success. In recent months the child began to complain of occasional frontal and temporal headaches, which were apparently not severe and which would subside after a few hours.

Examination at the time of the first visit revealed a well developed and nourished child, age 6 years, who was extremely bright and co-operative (especially when one considers the number of painful examinations and treatments to the nose which she had received). The relevant physical findings were limited to the nose and they were as follows: The right nasal chamber was within normal; the left was filled with a foul smelling,* seropurulent discharge and on removal of some of it by suction, the mucous membrane and turbinates were seen to be swollen and somewhat inflamed. Transillumination of the right maxillary sinus was good; that of the left was poor.

X-rays of the sinuses (see Fig. 1) were taken and a flattened circular opaque shadow was seen, which looked like and subsequently proved to be a metal button (see Fig. 2).

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*It is interesting that the foul odor was not mentioned by the child or by the mother, except after careful questioning, nor could they fix the time when it began. This happens often in such patients since those in constant association with the odor become inured to it and do not notice it at first because of its gradual onset and slow progression.

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On June 2, 1938, under general anesthesia, the button was removed from the nose. It was wedged between the septum and the posterior tip of the inferior turbinate, the upper part of it apparently resting in the middle meatus. It was found to be slightly rusted and eroded, and the crevices filled with old blood clots and much debris. That there was only

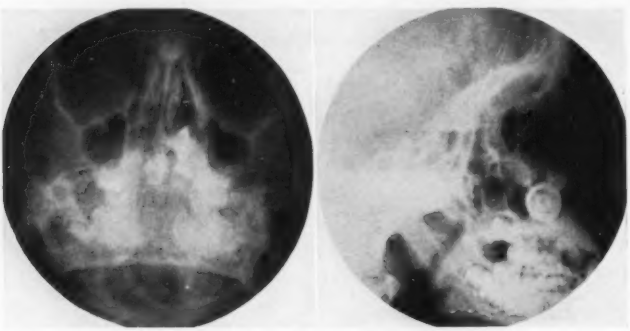

Fig. 1. X-ray showing foreign body in nose.

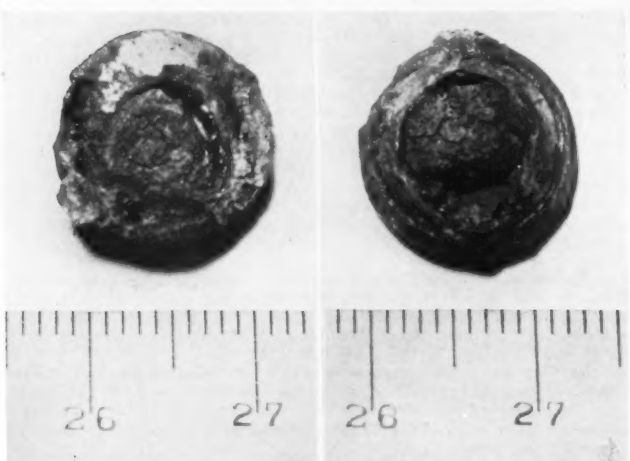

Fig. 2. Foreign body (metal button) in nose; diameter, 1.3 cm.; anterior and posterior views.

slight rusting may be accounted for by the fact that the button was of galvanized tin.

One week later the discharge had almost entirely disappeared, and with it the foul odor. Examination showed a slight inflammation in the region of the inferior turbinate. The child felt perfectly healthy. Re-examination

in six weeks showed no subjective or objective evidence that there had been any abnormality at all.

Summary and Comment: The case of a child with foreign body in the nose unrecognized for three years is reported. It should be emphasized that:

1. The presence of a unilateral rhinitis with purulent discharge, associated with a foul odor should always cause one to suspect the presence of a foreign body in the nose.

2. X-rays should be taken in all cases that do not respond to the average treatment.

79 Argyle Road.

750 St. Marks Avenue.

LINGUAL TONSILLITIS.

DR. HAROLD LIGGETT, New York.

The paucity of literature on this common adenopathy is surprising. In view of the many cases seen by the author, in addition to other cases acknowledged by personal communication from his colleagues, medical articles are indeed rare. Observation over a period of years establishes the conclusion that even in this day of super-specialism, neglect to use the laryngeal mirror for scanning the anterior pharyngeal wall and hypopharynx in searching for pathological lesions arouses amazement. The practice and skill required to observe the larynx is not necessary for viewing the lingual tonsillar region. Medical students are quickly capable of giving a good description of their findings with only a little practice.

I have seen a great many patients with "neuroses" of the pharynx; that is, patients complaining of the sensory symptoms of irritation, scratching, tickling, having pain in the throat, constant desire to swallow, lump in throat, irritable cough; and not a few of them suffer from disease of the lingual tonsil.

A physician consulted for a throat complaint cannot lay the blame on pharyngeal neuroses in instances wherein no pathology is seen in the pharynx. The patient who has had this affliction over a period of weeks or months, and localizes the symptoms to the hyoid region, should have special attention directed behind the tongue. It will be gratifying to both physician and patient to be told of the presence of large glandular masses — inflamed or even pouring pus from their follicles.

A chronic, persistent cough of a dry, harsh character, unexplained by the internist and not responding to chest therapy is one of the most usual results of lingual tonsil irritation.

In this paper I will endeavor to present several cases, histories typical of the lingual tonsil symptom-complex. These are representative of 50 cases observed and operated upon. They do not include cases which were medicinally treated by

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local application, nor others in which faucial (palatine) tonsillitis was prominent in the symptomatology.

When both groups in the Waldeyer chain seem pathologically deranged there is a nicety of judgment necessary on the part of the surgeon. Whether to operate both sets or pick out that group which gives the more evident disturbance requires a decision dependent upon several factors. Certain facts may accelerate the surgeon's judgment. If the patient has a general narcosis, as with ether, one may as well do both to obviate repetition of the anesthesia and expense. If there are extracapsular (palatine) tonsils bridging the gap between the lingual and the faucial tonsils, one must do the linguals also. Here, the excavation of the palatine tonsils leaves a depression which is overhung and permits these tags to grow into the faucial spaces. This will precipitate a so-called secondary growth of tonsil tissue, or what is believed by the laity (including many physicians) to be recurrences.

If the evident pathology is in the lingual tonsils and the palatine group is decidedly not infected, I do the lingual tonsil only. This is an unusual finding, as it seems that lingual tonsil disturbances make their appearance in persons who have had tonsillectomies performed. It may well be that the latter individuals had operations to relieve symptoms caused not by the palatine but by the lingual tonsil.

Lingual tonsils are the ventral segment of Waldeyer's ring of lymph glands. They are not always present microscopically. Often they are confused with the extracapsular projection of the palatine tonsils. This is not a true lingual tonsil, although they seem to bunch behind the tonsil laterally. Close observation will show that a goodly part of this mass lies in the tonsillar fossa and is often nothing but a postoperative hypertrophy of lymphoid tissue.

The lingual tonsil is a definite lymphatic mass which covers the posterior one-third of the tongue, extending laterally occasionally to the palatine tonsil and often almost touching the epiglottis. Posteriorly, it extends anteriorly to the circumvallate papillae.

The linguals have their own capsules, consist of two masses and are near the median raphe of the tongue. The glossoepiglottic ligament then divides them anatomically into a right

and left mass. They are usually confluent here, due often to an overgrowth of nodules, which to the eye gives a singular rather than a bilateral character. These nodules can extend above the tongue, making them apparent to the observer by only a slight pressure with a tongue depressor. The lingual mass does not often fill the vallecular space. When it does, the symptom of "fullness of the throat," or, in the presence of inflammation, the symptom of hoarseness is the complaint. When the nodules touch the epiglottis, the irritation set up by a more or less inflammatory disturbance causes a cough.

The mass of tissue is irregular and often a bunch of nodules will be more prominent on one side. Symptomatically, the patient will complain of disturbance on that side of the neck.

The lymphatic supply is limited and is drained by the group of glands along the internal jugular vein. The blood supply is particularly abundant, varices are commonly observed — there is, however, no problem of bleeding in surgery of the lingual tonsil, nor are there any septicemia or pyemic cases reported from disease of the gland *per se*.

Local infection, that is, acute inflammatory conditions at the base of the tongue, occur quite commonly, however. They are very severe and dangerous, causing deep abscesses in the tongue (lingual quinsy), situated beneath the capsule which separates the lingual tonsil from the deeper structure of the tongue. Often one sees cases of Ludwig's angina. In the latter, the infection follows the fascial planes of the neck. The former is a deep-seated, localized process. Suppuration in this area is not to be regarded too lightly, but treated as a major surgical problem because of the involvement of the epiglottic-arytenoid fold, with possibly a subsequent laryngeal edema. Also, the increased blood supply makes amateurish stabbing and incising a risky procedure.

Microscopically, the lingual tonsil cannot be distinguished from the faucial tonsil; there are, however, certain characteristics which distinguish it as a separate organ. The capsule of the lingual tonsil is not as definite a structure as is seen about the palatine tonsil. The capsule is a fibrous covering, which does not enclose the lingual tonsil as it does its neighbor. The capsule is a thin tissue which acts as a line of demarcation from the underlying muscle structure of the tongue.

The crypts of the follicles of the lingual tonsil are not so numerous as those of the palatine tonsil. They are shallow. They run only in an anteroposterior direction; that is, parallel to the direction of the tongue. They are not deep and thus usually empty of debris and secretion. (It is difficult to express any secretion, due probably to the inability to exert sufficient pressure against the tongue.) Crypts, here, are comparatively larger than the crypts of the palatine tonsil, and this makes them more liable to infection. Thus, a postnasal drip will occasion an infection in this region, whereas the hypopharynx and larynx are rarely thus directly involved. The width and

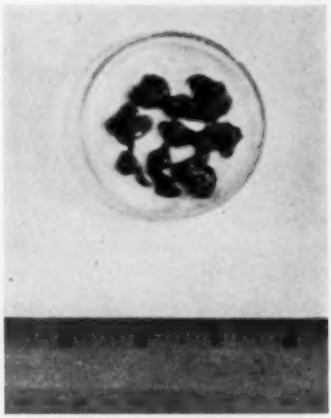


Fig. 1. Lingual tonsil tissue removed from Case E. R. Scale indicates actual size.

shallowness of the lingual crypts does not permit absorption over too lengthy a period, so that patients with focal symptoms are not perennially ill. The length of time that symptoms of lingual tonsilitis lasts varies from one week to 10 weeks. This latter fact will help distinguish palatine tonsillitis from infection of the lingual tonsils.

One will find here all the diseases of the Waldeyer lymph chain, acute and chronic inflammation, keratosis, but the most common one, as in the fauces, is hypertrophy. This is important, not because of its inflammatory significance but on account of its size. The lingual tonsil may protrude into the pharynx sufficiently to arrest a sharp morsel of food.

A fish bone on its accidental journey to the stomach will scratch the lingual tonsil and set up an intense local inflammatory reaction. In some individuals (of neurotic temperament) who insist a bone is stuck in the throat and who will definitely localize it, I use the following method to reassure them: I paint the area pointed out with cocaine or silver nitrate. This, of course, relieves the sensation. When they swallow in order to discover how successful I've been, I tell them that they may feel the pain on the other side or in a different location. This anticipates their doubt as to my diagnosis. I then proceed to apply silver nitrate, which is almost a specific.

Ordinary tonsillitis (palatine) gives symptoms from two days to one week; never more than two weeks. It follows, then, that infections derived from lingual tonsillitis are not as great or as severe, though they last longer than infections which are in the palatine tonsils.

Inflammations of the lingual tonsil are parenchymatous in nature, the entire gland becoming red and hypertrophied. Hypertrophy is indicative of increased activity, so that in chronic lesions we must suspect this wide area to be the seat of focal infections. Acute follicular inflammations are uncommon; however, due to the chronicity of the lesions, a keratotic degeneration (mycosis) is common. The hard, gray pieces are often seen behind the tongue — each keratotic deposit extends from or lies upon a follicle or crypt and is especially difficult to remove.

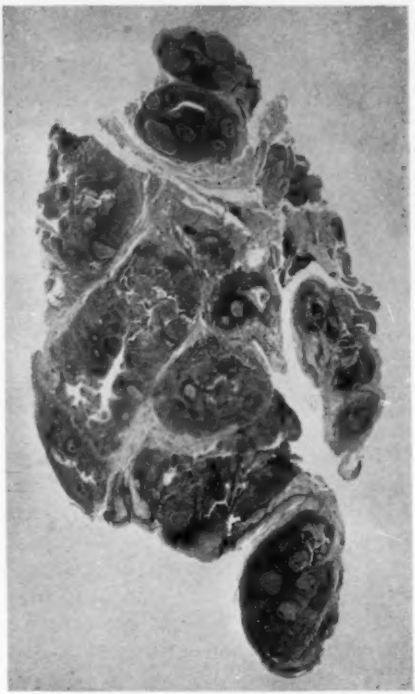
A typical pathological report of a microscopical section is described and illustrated.

In the main, patients go a long time without relief from disturbances of the lingual tonsils. Eventually, however, when all casual exurberances have been fulgurated, cauterized or operated upon, the patient, in desperation, submits himself or herself to the skilled treatment of a well trained laryngologist. The history is typical. Our laryngologist makes a careful examination and finds the etiological factor.

Lingual tonsils, when pathologically disturbed, give a definite history and symptoms which are an immediate clue to make us suspicious of their presence.

Feeling of a lump or swelling with occasionally a dull ache in the throat is the most common complaint. Women who come

for relief suspect the thyroid gland. They have a suspicion that the thyroid is growing. The ache or pain is usually described as a sort of tightness or heaviness. The sensation is localized only by an effort and some thought to be in the front of the throat, "deep inside." Patients point to a wide area in the region of the hyoid bone.



Lingual tonsil, microscopic section, low power.

Palatine tonsils never give this localization. The patients point inside their mouths or show the pain to be on the side of the neck. The latter region is also the location of the lymph glands draining the fauces. The interesting feature of the sore throat from lingual tonsils is the duration of the symptoms. This fact is diagnostic. When treatment which is applied to the fauces and the postpharyngeal wall is not effica-

cious, attention should be directed to the lingual region. Other symptoms, as a dry, constant cough, tickling sensation, especially at night, a tender and painful area behind the clavicle, are important but not conclusive. All symptoms are most marked in neurotic individuals.

Even in the presence of infected faucial tonsils, no sore throat could be due to anything else. Were one to ascribe this infirmity to the faucial tonsil, it would be well to remember that pain and soreness of faucial tonsils is remittent and that attacks, even when they begin with a severe follicular or parenchymatous inflammation, do not last more than a week. Of course, one must distinguish acute lesions, such as ulcerative tonsillitis, cellulitis or peritonsillar abscess formations. The latter are quite obvious. Pharyngeal "neuroses" may present some difficulty, but the localization of burning, tickling, scratching, choking, hair sensation, are confined to the posterior pharyngeal wall and are immediately relieved by an application of $AGNO_3$. Hyoid bursitis is rarely encountered and easily differentiated.

The enormity of the discomfort produced by lingual tonsil disease in the mind of the patient, makes him a bad subject for treatment. The condition will yield very quickly to proper therapy. Complications and unpleasant sequelae never or seldom occur.

The treatment is medical or surgical. The latter alone will establish a cure or cessation of symptoms, while medical treatment will give relief. The topical administration of silver nitrate (20 to 30 per cent) is very efficacious in acute lesions. Any astringent medication, such as tincture ferric chloride or iodine in glycerine, will be of benefit. Caustics, such as trichloracetic acid, are good. The (actual) galvanic cautery or diathermy is a good way to destroy the gland. Except in the presence of lingual varices, there is no contraindication to surgery; however, varices can easily be seen and so avoided.

I find the lingual tonsillotome is most satisfactory. This is a guillotine type of instrument, devised by Myles, which successfully excises the lingual tonsil level with the tongue. The instrument is equipped with teeth which grasp the tissue and prevent aspiration. Cocaine acts as a very good astringent, contracting the blood vessels and at the same time anesthetiz-

ing the area. This is all done by indirect laryngoscopy. It may be done with the patient in a semi-reclining position. In a few minutes the tonsillotome is applied and reapplied until the tongue surface is smooth. Bleeding is negligible. The patient, pulling his tongue, is soon able to co-operate with the surgeon. The latter operates between "spits," as it were. If the bleeding obscures the field, wiping the area, at the same time applying gentle pressure, will soon help visibility. (Note: I suggest the use of running hot water for the laryngeal mirror. It will heat the glass and at the same time wash the blood away. Since nearly all laryngologists place the patient's

Lingual tonsil, microscopic section.

chair next to a sink, this suggestion may easily be followed.) There is no reaction following the operation, nor any after-pain or postoperative care or discomfort. Patients may go home. A bland, warm gargle and a throat lozenge will prevent or diminish bad breath. In a few days upon return to the office, the doctor cleanses the area of sloughs and applies $AGNO_3$, 5 or 20 per cent, when necessary.

The majority of tonsillectomies are not performed by the laryngologist but rather by the general practitioner. I hold no brief for the exact partition of "specialties." A general practitioner or general surgeon may be able to do a tonsil-

lectomy equally as well as a trained laryngologist, but if he applies his surgical skill in this direction, let him be sure that the throat symptoms are not nerves, sinuses, adenoids or tonsils. They may be caused by lingual tonsils, and his discomfiture when confronted by an unrelieved and angry patient could well have been avoided.

Miss E. R., colored, age 31 years, was advised to have her tonsils removed three years ago for repeated attacks of sore throat, with typical localization referring to the hypopharynx.

Upon medical advice, she deferred this procedure, due to uncertainty as to the relationship of the lingual tonsils to these symptoms. Sore throat continued, and the patient began to experience "sticky" pains in the neck, radiating downward from the thyroid region to the root of the neck.

About one and one-half years ago, arthritis developed. She had pains in the joints of her fingers, pains in her legs and, generally, all her joints ached. There was no swelling of the joints. One year ago, the "sticky" pain in the neck spread over the chest and was always associated with sore throat. The latter condition she described as a dull pain inside and deep in the throat. These attacks lasted three to four days and recurred at intervals of one to two months.

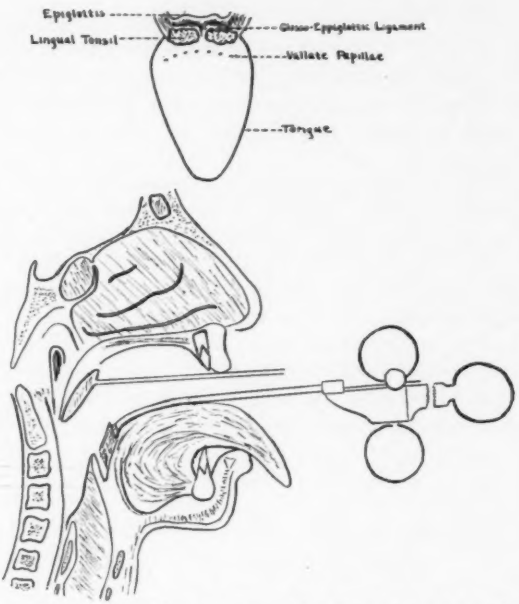
Medication and various therapeutic measures having failed to cure her, she returned for an operation upon the lingual tonsils.

Examination at this time revealed hypertrophied lymph nodules spread over the entire vallecula and extending anteriorly over the base of the tongue, so that the glands could be seen even without a reflecting mirror. The lingual tonsils were very nodular and cryptic and in a state of subacute inflammation. The pharynx otherwise was entirely normal. There were no faucial tonsil remnants and no postpharyngeal lymph granules.

She was operated upon and a very large amount of lingual tonsillar tissue was excised. The postoperative course was uneventful. She was free of all symptoms within one month. It is seven months since the operation at this writing, and the patient has had no more sore throat, no paresthesias and no more joint pains.

I consider this case to be a classical example of the relationship of lingual tonsil disease as a focus of infection for systemic involvement with eradication of the focus removing the symptoms.

Case 1: Miss A., age 10 years. This girl was suffering with an acute attack of laryngeal stridor. Her tonsils had been



Above — Sketch indicating hypertrophied lingual tonsil with adjacent landmarks.

Below—Mirror and lingual tonsillotome in position for operation.

removed several years ago, in spite of which she had frequent cold and occasional sore throat.

For the past two or three years her colds gave a peculiar hacking cough, nonproductive, which persisted for eight to 10 weeks in each attack.

First examination proved negative for tuberculosis and asthma, and there was no evidence of bronchitis. The cough was accompanied and apparently stimulated by an irritating "something" in the throat.

The present attack of croup began with a slight sore throat, with temperature, 24 hours previous.

Pharyngeal and laryngeal examination revealed no membrane, but seated behind the tongue and at its base was a large hypertrophied infected lingual tonsil, about 2 cm. in diameter. It was cryptic; some of the follicles exuded pus and others presented capillary markings.

This patient was operated upon several weeks later and the lingual tonsil removed. It is now 15 months since her operation and there have been no coughing spells nor any irritating fullness in the pharynx, in spite of having had one severe upper respiratory infection.

Case Mrs. B., age 42 years: Every year, following exposure, she developed an upper respiratory infection, which manifested itself by an irritable throat and later a "fullness of the throat," with a dull ache, localizing itself "back in the front of the neck." (She points to the suprahyoid region.) Accompanying this symptomatology, she has a chronic, dry, hacking cough, nonproductive in character and usually started by a tickling in the throat. More significant and of importance to the theory of focal infection is the onset of pain in the left hip joint. Her lingual tonsils, which were hypertrophied and nodular, were excised. Recovery was complete. Has had no attack for two years.

Case Mr. J., age 41 years: Has had a fullness and aching sensation in the throat for many years. No diagnosis was ever made aside from pharyngitis due to a postnasal drip. He was treated for hypothyroidism and anemia. The fullness and pain persisted, even at times when the postpharyngeal wall looked normal, and was so diagnosed by his attending physician (a throat specialist). The above symptoms were disregarded, in spite of numerous consultations to explain a general feeling of malaise; more specifically, a pain in the left shoulder and neck muscle, occasionally occurring on the right side. There was arthritis of both knees. Faucial tonsils had been removed — there was no secondary growth. The lingual tonsils were hypertrophied, consisted of nodules, which on the left side stood out from the tongue more prominently than on the right side. During periods when symptoms were aggravated, an inflammatory mass could be easily detected. He was

operated upon several years later. The symptoms subsided promptly and recovery is very complete.

Case M. B. C., age 29 years: Had sore throat, lasting a few weeks, once or twice a year. On advice of the family physician, the tonsils were removed, and attacks continued, with no relief from symptom. Examination showed presence of lingual tonsils. Operation gave complete recovery.

Case A. L., age 31 years: Had annoying sore throat. Never had fever, but the condition made her nervous. Always refused a tonsillectomy. Finally came to me for removal of tonsils. Examination showed apparently normal faucial tonsils buried in the pillars. No pus was obtained on expression. The anterior pillars were not congested. The lingual tonsils were hypertrophied and cryptic. Application of $AGNO_3$ to the lingual tonsil gave relief of symptoms. This was repeated for several days. Note: I might say that this procedure is a definite therapeutic test, proving the infection to be localized in the lingual tonsils.) She was operated upon a few weeks later, with complete recovery.

Case B. S., age 37 years: Had sore throat for two weeks. Tonsils had been removed a few years ago. Examination showed presence of large number of gray-white, hard nodular concretions scattered throughout the pharynx but mainly centered in the lingual region upon tremendously hypertrophied lingual tonsils. The majority of the keratotic spots were removed fairly easily from the fauces and postpharyngeal wall. In the lingual region it was deemed advisable to excise them *en masse*.

A lingual tonsillectomy was done. Following several days of treatment, when occasional nodules were removed in the surrounding area, the patient had an uneventful recovery.

Case M. B., age 13 years: Was brought to me for dysphagia and impaired speech. He had a sore throat for three days, following which the above symptoms developed. His doctor noted nothing unusual in the pharynx except possibly some redness. The tonsils were embedded and no inflammation was apparent. Examination showed a hard, board-like tongue posteriorly, by palpation. Mirror examination showed a raised inflammatory area over the left tongue base. No confluency of tissue marked off a lingual tonsil, but surrounding this area

and in the entire vallecular region were small, raised lymph nodules. Slight pressure on the tongue revealed a discharge of pus througn the inflamed follicle.

Diagnosis: Lingual abscess.

Treatment: The fistula was dilated and considerable pus evacuated. The patient made an uneventful recovery. Subsequently, a tonsillectomy under general anesthesia and removal of excess lingual tissue was done. The patient has had no recurrences.

Case Mrs. L., age 36 years: Was referred for examination of the pharynx to explain a chronic, annoying sore throat. For two years she had been bothered by a heavy sensation; a sense of pulling in the pharynx. There seems to be something at the back of the tongue. There has been a series of attacks lasting two or more weeks, with remissions of several months.

The condition has been diagnosed as due to stumps of tonsil tissue, remnants of a tonsillectomy done 14 years ago. There have been several disagreements among her physicians as to the etiological factor.

Examination showed small tabs of tonsil tissue at the base of the tonsillar fossae. These tabs are normal in appearance. The fossae above are clean and well excavated. Behind the tongue and filling the valleculae are large masses of lingual tonsils. An application of $AGNO_3$ directed to the latter gave relief of symptoms, so that she was averse to having an operation. She said $AGNO_3$ applied to her throat several times before gave no relief. This definitely localized the lesion.

Operation performed. There has been a complete cessation of symptoms for 15 months.

Conclusions: The diagnosis of globus hystericus and of pharyngeal neuroses has often been mistaken for lingual disease.

In adults, lingual tonsil infection should never be overlooked in explaining pharyngeal or throat symptoms. If the faucial tonsils are removed, patients should be advised of the presence of lingual glands which may give trouble in the future.

Microscopic Description: Case Mr. J.: Sections of lingual tonsils show squamous epithelium, with some superficial keratinization, and underlying compact lymphoid stroma, with some active follicles. Deep crypts contain some amorphous content and show vacuolization of epithelium and cell infiltration. In some areas the epithelium is atrophic, and in others definitely hyperplastic. Underlying vessels show cellular engorgement. In the crypts some amorphous debris can be made out. No purulent exudate; fairly extensive inflammatory infiltration.

Diagnosis: Lingual tonsillitis; marked lymphoid hyperplasia.

133 East 58th Street.

OBSTRUCTIVE LARYNGEAL DYSPNEA.*

DR. SAMUEL WEINSTEIN, Brooklyn.

The otolaryngologist is frequently confronted with obstructive laryngeal dyspnea. It may present itself with such speed and intensity that immediate action may be necessary to prevent asphyxiation, or it may progress slowly, and so allow time for careful diagnosis and decision as to the type of treatment. This paper was prompted by the fact that within a short period of time I was confronted by three cases of obstructive laryngeal dyspnea, two of which terminated fatally.

Obstructive laryngeal dyspnea is a very serious symptom of a number of conditions and may require emergency relief so frequently that it remains a constant problem to all practitioners. Since the dyspnea may be urgent, every physician should be able to determine whether the obstruction with which he is confronted requires relief by tracheotomy, and, furthermore, he should be able to perform the tracheotomy if necessary.

Cassidy[1] quotes Gabriel Tucker as defining obstructive dyspnea as dyspnea due to obstruction in the airway, and as classifying it very aptly into three types: *1.* high obstructive dyspnea when the etiology is found in the pharynx, larynx or cervical trachea; *2.* low obstructive dyspnea when the lesion is in the thoracic trachea or main bronchi; *3.* bronchial obstructive dyspnea when the obstruction lies in the small bronchi.

The cases described in this paper fall within type I of the above classification.

CASE REPORTS.

Case 1: No. 209,027, W. R., white, male, age 3 months. Diagnosis: Foreign body in larynx. Asphyxiation.

This infant, having a bilateral harelip and cleft palate, was brought to the hospital by the mother, who said that it had "swallowed" the nipple from a Brecht feeder about five minutes before, and that it was having difficulty in breathing.

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*From the Otolaryngological Service of Dr. Philip Leibowitz, Jewish Hospital, Brooklyn.

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In the elevator, on the way to the bronchoscopy room, it had taken a deep gasp and ceased breathing. Artificial respiration was immediately instituted. An apparently lifeless child was placed on the bronchoscopy table and the nipple immediately removed. It had been lying horizontally across the lumen of the larynx.

Artificial respiration was continued during this entire procedure, and, in addition, after the nipple was extracted a bronchoscope was passed and oxygen administered through it. The infant was also given an ampoule each of coramine and alphalobelin, and an intracardiac injection of adrenalin, MX.

In spite of all these measures, the child did not begin to breathe, and as no heart beat could be obtained, after about 15 minutes the child was pronounced dead. It apparently had died in the elevator.

Pathology Report: The specimen is a soft rubber nipple, 4 cm. long and up to 1.3 cm. in width.

Case 2: No. 210,348, R. S., white, female, age 3 years. Diagnosis: Streptococcic laryngotracheitis. Edema of larynx.

This patient was admitted to the Jewish Hospital on March 22, 1938. The night before, the child had been restless. On awakening at 8 A.M., she appeared to have some difficulty in breathing and a change in her voice was noted. She complained of pain in the lips, but not in the throat. Her temperature at that time was 101.5° F.

A physician was called, who thought that the child had either a retropharyngeal abscess or a laryngotracheitis, and he advised hospitalization.

The patient was admitted to the hospital in acute obstructive dyspnea, with marked difficulty in inspiration, cyanosis, suprasternal, supraclavicular, infraclavicular, intercostal and infrasternal retraction. Mirror laryngoscopy revealed a red, edematous epiglottis, which overlay the entire glottis.

Laboratory Reports: Urine, negative. Blood count on admission, March 22: Hemoglobin, 73 per cent; red blood cells, 5,400,000; white blood cells, 47,200; polymorphonuclears, 95 jer cent; lymphocytes, 3 per cent; metamyelocytes, 2 per cent; band cells, 16. Throat and nose culture, no pathogenic organisms. Culture taken from larynx, negative for Klebs-Loeffler bacillus.

A low tracheotomy was performed, in the ward, under local anesthesia, and the dyspnea was immediately relieved. Direct laryngoscopy, several hours later, with the Flagg laryngoscope showed the epiglottis, arytenoids, aryepiglottic folds and false cords to be edematous and red. The true cords could not be seen. No membrane was seen.

Consultation with the pediatric service revealed no other cause for the dyspnea.

The postoperative course was uneventful. The child was given special nursing, day and night, with the usual postoperative care of the tracheotomy tube, suction machine at the bedside and steam inhalations. She was put on sulfanilamide gr. X q. 4 h. and sodium bicarbonate gr. X q. 4 h.

The child's temperature the day after the operation was 103° F.; it then hovered around 101° F. until the sixth postoperative day, when it reached 99° F., and on the eighth day it was normal.

The tracheotomy tube was removed four days postoperatively, the sulfanilamide was discontinued on the sixth day, and on the twelfth day, April 2, 1938, the child was discharged. On March 28, mirror

laryngoscopy revealed no edema and the cords were visualized. These were slightly pinkish, otherwise negative.

Case III: No. 210,528, A. M., white, female, housewife, born in Russia, age 52 years. Diagnosis: Pneumonia, agranulocytosis, edema of larynx.

This patient was admitted to the Jewish Hospital on the medical service of Dr. J. Rosenthal on March 29, 1938, with the history of having had bronchopneumonia five weeks previously and of having been fever-free for two and one-half weeks. About 24 hours before admission, she had a tooth-chattering chill, temperature of 102.5° F. and a sore throat. Her physician advised hospitalization for blood tests.

At the hospital, it was definitely established that she had been taking sulfanilamide tablets since the onset of her illness, three pills t.i.d. for four days, and then one pill t.i.d. until the day before admission. The exact dosage was not ascertained.

On admission she appeared acutely ill, the face was flushed, the tongue was slightly coated, the pharynx and uvula were reddened and covered with a whitish exudate. There were dullness, moist rales and impaired resonance, with increased tactile fremitus over the base of the right lung.

The blood count on the day of admission was: Hemoglobin, 85 per cent; red blood cells, 4,700,000; white blood cells, 1,500; lymphocytes, 96 per cent; mononuclears, 4 per cent.

The diagnosis on admission was agranulocytosis and pneumonia, right lower lobe.

The treatment instituted was small doses of X-ray to the flat and long bones, yellow bone marrow extract m. 50 t.i.d., high vitamin and high caloric diet, and forcing of fluids.

The patient's temperature throughout her stay in the hospital was between 105° F. and 105.4° F.

On the afternoon of March 31, 1938, the medical service noted that the patient had an obstructive type of breathing and a nose and throat consultation was ordered.

The patient looked flushed and her respirations were noisy, but she did not seem to be in any immediate danger, and there was only a very slight retraction in the left supraclavicular fossa.

Laboratory Reports: Blood examined for Heterophile Agglutination: Agglutination occurred in the dilutions up to 1:32, inclusive (normal limit, 1:32). Sputum typing: Pneumococcus type VI. Blood culture, negative. Kline test, negative. Urine, negative. Blood counts: March 29, 1938: See above. March 30, 1938: Hemoglobin, 81 per cent; R.B.C., 4,300,000; W.B.C., 1,000; lymph., 100 per cent. March 31, 1938: Hemoglobin, 81 per cent; R.B.C., 3,700,000; W.B.C., 800; lymph., 100 per cent.

Indirect laryngoscopy in the ward was unsuccessful, and as the patient's soft palate and pharynx were covered with exudate, it was thought that there might be a similar condition in the larynx; therefore, she was taken to the operating room for direct laryngoscopy and suction, or possible tracheotomy.

As laryngoscopy was attempted she struggled and insisted that she would not allow anything to be done. It was then deemed advisable, in spite of her protestations, to perform a tracheotomy.

As she was being prepared she suddenly became cyanotic and ceased to breathe. Artificial respiration was immediately instituted, a cricothyrotomy was performed, which was soon followed by a low trache-

otomy, and oxygen, intracardiac adrenalin and coramine were administered. In spite of all these measures the patient did not respond.

Anatomic Diagnosis (Autopsy): Primary: Wound of recent tracheotomy, gangrenous pharyngitis, tonsillitis, edema of larynx, arteriosclerosis, generalized; nephrosclerosis, hypertrophy and dilatation of heart, passive congestion of viscera. Subsidiary: Cysts of kidneys, cyst of thyroid gland, fibrous peritoneal adhesions.

COMMENT.

Impaction of a foreign body in the air passage is a potentially dangerous event, which always calls for cool judgment on the part of the medical practitioner and occasionally for an unparalleled rapidity of action. The first case was one of inspired foreign body in an infant, in which, had immediate digital palpation of the hypopharynx been attempted there would, no doubt, have been a successful removal of the nipple. Certainly, tracheotomy or emergency tracheotomy, performed as soon as the obstruction was recognized, would have saved the infant's life. To ensure the proper handling of such cases all hospitals should deem it necessary to have internes in charge of the emergency ward trained in the performance of tracheotomy.

Crow[2] says that in the air passages it is the bulky, rounded object which is apt to be urgent, and not the thin, sharp object, as in the food passages. It is seldom that the patient with a really obstructive foreign body survives long enough to reach a medical man, but if he does so, a clear line of action is imperative: *1.* invert the patient; *2.* try to hook out the object from the larynx with the finger; *3.* incise the cricothyroid membrane. In such an extreme case of danger there is no time to consult references on surgical anatomy, and it is, therefore, essential that every medical man be familiar with the cricothyroid space.

Case 2 was one of streptococcic laryngotracheitis, in a child, with edema of the larynx resulting in acute obstructive dyspnea. The seriousness of the obstruction was immediately recognized, and a successful termination followed tracheotomy, the usual postoperative nursing care, steam inhalations, alkalies and sulfanilamide therapy.

Laryngotracheitis with obstructive breathing is not rare or new, but it is often not recognized and is not infrequently treated as diphtheria, foreign body or pneumonia. It is chiefly limited to infants and very young children. The most preva-

lent age is from one to three years. It is possible that older children may be afflicted with this condition, but the structures involved are less vulnerable to swelling and obstruction.

Gatewood[3] says the mortality rate is very high, varying with different observers from 35 to 70 per cent. When bronchial obstruction dominates the picture, the condition is especially serious. If the obstruction is mainly laryngeal and there is no association with atelectasis, mechanical relief is reasonably certain, and the prognosis is good. He says that bronchial asthma, spasmodic croup, laryngeal edema, thymic pressure (if there be such a disease), diphtheria, foreign body and bronchopneumonia should be considered when examining cases with obstructive breathing.

Smith,[4] quoting Richards, says that laryngotracheitis should always be thought of with two other important clinical entities, *viz.*, *1.* diphtheria, and *2.* a foreign body. Even in the face of an epidemic, these two conditions should always be ruled out.

In a study of 43 cases, he found that the condition was accompanied by some signs of an infection of the upper respiratory tract. Many of the patients have only a mild pharyngitis or rhinitis for 24 to 48 hours preceding the onset of laryngeal involvement.

Kelly[5] says that, after seeing a few of these cases, it is easy to become panicky over a case of croup, and he believes that a child with a persistent croup and a temperature of 101° F. should be hospitalized so that proper treatment may be instituted.

Case 3 was apparently one of agranulocytosis, due to sulfanilamide, complicated by edema of the larynx. The obstructive laryngeal dyspnea was recognized and treatment attempted. Suffice it to say here that sulfanilamide may cause agranulocytosis[6, 7, 8] and that agranulocytic angina may involve the larynx.[9] The form of involvement may be inflammatory edema, ulceration and necrosis of laryngeal structures in various parts.

Agranulocytic angina usually occurs in women of middle age, and when the white cell count drops to 1,000 cells per cmm. or less, the outcome is almost invariably fatal. It was suspected that this patient might have a similar exudate in

the larynx as on the pharyngeal wall and soft palate and, therefore, direct laryngoscopy and suction were attempted.

Kully,[10] in speaking of laryngeal obstruction in children, says that aspiration cannot relieve the edema of the subglottic space, which is the most common form of obstruction in children, and the increased respiratory effort resulting may precipitate a laryngeal spasm with complete obstruction. This may be brought on by severe exhaustion, associated with gagging during laryngeal examination. This mechanism may have been the cause for the sudden cessation of respiration in this case, although not a child. Of course, it must be remembered that this patient was extremely toxic, with a high temperature, pneumonia, 800 white cells in the blood, and that her condition was, no doubt, a terminal manifestation.

Davis,[11] in discussing the various types of acute laryngeal obstruction, considers the most dangerous type of all as that of acute septic laryngitis accompanying pneumonia and bronchopneumonia in elderly patients. The sudden occurrence of heart failure may convert a moderate edema into a complete obstruction, which requires an immediate tracheotomy. Even if a timely tracheotomy is performed, the patient may succumb to heart failure and severe toxemia.

It will be recalled that at the autopsy of this case there was found a congestion of the viscera. Dart,[12] in a study of seven cases of sudden death from asphyxia, says that the associated diagnosis of generalized passive congestion, especially of the lungs, engorged right heart and vena cava, and small ecchymoses on the surfaces of the pleurae, pericardium and peritoneum are fairly constant findings in all deaths from asphyxia.

DISCUSSION.

Platou and Hilleboe,[13] in an excellent study of obstructive laryngitis, classify their nondiphtheritic cases as follows:

1. Acute Nondiphtheritic Laryngitis: Cases with obstructive laryngeal symptoms, but lacking a history of gradual progression and hoarseness, make up this group. They are characterized by an accumulation of mucus in the throat, redness and edema of the larynx and absence of laryngoscopic or bacteriologic evidence of diphtheria. They are often asso-

ciated with simple rhinopharyngitis. A synonym for the term suggested is acute catarrhal laryngitis.

2. Acute Exudative Laryngotracheitis: Cases with obstructive laryngeal symptoms, accompanied by rather profound constitutional symptoms and characterized by redness, edema, purulent or fibrinous exudate and "moss agate" plugs below the cords, but with no evidence of diphtheria, make up this group. This form is not infrequently associated with influenza, measles and scarlet fever.

3. Miscellaneous Group: In this group are included such conditions as retropharyngeal abscess, foreign body, tumor, asthma, angioneurotic edema, tuberculosis and syphilis.

The chief symptoms of obstructive laryngeal dyspnea, according to the Jacksons,[14] are restlessness, increasing respiratory rate, stridor, indrawing at the suprasternal notch and ashy-gray pallor. The locations of indrawing are: *1.* the suprasternal notch; *2.* the supraclavicular fossa; *3.* the intercostal spaces; and *4.* the epigastrium.

The most important of these is the indrawing at the suprasternal fossa; next is the epigastric indrawing, especially in children.

The indrawing at the suprasternal notch is diagnostic of obstructive laryngeal dyspnea. It is never seen in the dyspnea of asthma, pneumonia or other diseases, mediastinal, cardiac or pulmonary, unless laryngeal complications are present, and in urgent cases tracheotomy should be done first on this sign alone; the diagnosis can be completed afterward.

Two other important signs accompany this indrawing; namely, stridor and restlessness, but they are not absolutely diagnostic of obstructive laryngeal dyspnea. Restlessness is very important, as it is indicative of air-hunger.

Cyanosis is late, and may be absent to the end. Ashy-gray pallor is more diagnostic.

Laryngeal symptoms in children usually occur with a sudden onset and not insidiously, as in adults, and frequently become very urgent, due to anatomic conditions. Patterson,[15] in discussing laryngeal symptoms in children, says the glottic chink of a child with a normally developed larynx is small and readily encroached upon by a very slight amount of inflam-

mation or swelling of the laryngeal mucosa, thus interfering with respiration. The subglottic area in little children under age 3 years is very vascular, the connective tissue very loose; hence, swelling quickly results from inflammation, infection or trauma, and quickly becomes a factor in all cases of laryngeal disturbance.

The diagnosis as to the cause of dyspnea requires mirror laryngoscopy, direct laryngoscopy and bronchoscopy. Most important is the diagnosis of the location of the obstruction; that is, whether it is above or below the clavicle. The former, as mentioned previously, is termed obstructive laryngeal dyspnea, and the cardinal signs and symptoms have been discussed.

Silverman,[16] in a study of 2,500 hospital admissions over a period of five years (1929-1933), found that 44 cases of this group were admitted with laryngeal symptoms. These 44 cases, he found, could be arranged on an etiological basis as follows: diphtheria, 12; measles, three; congenital, two; catarrhal (croup), seven; laryngotracheitis, 20. One sees, therefore, that only 27 per cent of the cases were due to diphtheria, whereas the group known as laryngotracheitis or laryngotracheobronchitis comprised 45 per cent of the group.

Jackson[14] says that low tracheotomy is the only treatment in a case of well marked obstructive laryngeal dyspnea. He mentions three exceptions to this rule: *1.* If the case is one of laryngeal diphtheria, and there is a skilled intubator, who will not only introduce the intubation tube but will remain in the house to reintubate in case the tube is expelled, intubation is worth consideration; otherwise it is not. *2.* Laryngismus stridulus is accompanied by indrawing but the dyspnea may subside without tracheotomy. *3.* Retropharyngeal abscess may require only excavation.

Intubation and tracheotomy both prevent asphyxia, but tracheotomy has the further advantages of draining the secretions and putting the larynx at rest.

A patient with signs of beginning obstructive laryngeal dyspnea should immediately be removed to a hospital for continuous observation, and such a patient should never be given morphine or any other sedative, for he is depending on his voluntary respiratory muscles to draw in enough air, and under sedation he will lose the aid of this mechanism.

Smith[4] says that palliative treatment should only be considered in patients with mild involvement, in whom retraction is not present, or only mildly so and is not increasing. In the presence of toxemia such measures as a high intake of fluids, the administration of antipyretics and hydrotherapy are all employed. Inhalations of tincture of benzoin are used and the room is kept at a constant temperature of 76° to 78° F. Sedatives are never given.

In the more severe cases, surgical intervention is usually necessary, and tracheotomy is usually called for. He says that it is preferable to precede tracheotomy with bronchoscopy, leaving the bronchoscope in place. Tracheotomy can then be done with greater leisure and ease, because the trachea will be easily identified with the tube in it, and at the same time there is no asphyxiation.

I might recall here, in line with recent therapy, the use of sulfanilamide in indicated infective cases, as in Case 2 of this paper.

Kully,[10] in discussing the various methods of relief of obstructive laryngeal dyspnea in children, such as intubation tracheotomy and suction, says that "repeated suction through the laryngoscope or bronchoscope seems to have little place in the treatment of laryngeal obstruction in children."

In extremely urgent cases of edema of the larynx with heart failure, there may not be any time for any kind of preparation, and a stab laryngotomy is necessary to save life.

Laryngotomy is only performed as a temporary measure in cases of extreme urgency, as retention of the tube, after a few hours causes edema of the cricoid ring and injury to the larynx. It is, therefore, subsequently necessary to do a low tracheotomy.

Davis[11] says that if an intratracheal catheter is at hand with a direct laryngoscope, the larynx is quickly swabbed with cocaine and the catheter inserted through the larynx. This method of temporary intubation has the advantage of making a subsequent tracheotomy easier and less of a scramble.

I would add here that a simpler procedure, and often a life-saving measure until proper tracheotomy can be performed, is the insertion into the larynx of the Mosher, or similar type,

of life-saver tube. Every practitioner should carry such a set of tubes in his bag and should be skilled in their use.

Figi,[17] in a study of 200 cases of tracheotomy, said that, providing dyspnea of cardiac or pulmonary origin has not been mistaken for the obstructive type, and providing tracheotomy has not been too long delayed, complete relief from difficulty should follow the operation. Lack of relief after opening the trachea and the aspiration of the accumulated mucopurulent secretion is always a serious sign, for it means that either the diagnosis has been wrong, or that the tracheotomy has been too long delayed and cardiac failure or bronchopneumonia has set in.

In that series of cases bronchopneumonia was the most frequent postoperative complication. Mediastinitis was not encountered in any case.

The deaths in the series were almost entirely the result of dyspnea and delay in opening the trachea, rather than of the tracheotomy itself.

Carpenter,[18] discussing acute laryngeal stenosis in children, says that tracheotomy in itself carries a mortality of scarcely 1 per cent, but the occasions for this operation are serious and this is the reason for a high mortality.

The technique of tracheotomy I shall not discuss. I will simply mention that the after-care of the tracheotomized patient is extremely important. Every tracheotomized patient should have special nurses, day and night, who are especially trained in the care of such cases.

SUMMARY AND CONCLUSIONS.

1. Three cases have been described, each of which presented the symptom of obstructive laryngeal dyspnea, but in each of which the fundamental etiology was different; namely, *a.* hypopharyngeal foreign body in an infant, with occlusion of the laryngeal lumen; *b.* streptococcic laryngotracheitis in a child with edema of the larynx; and *c.* edema of the larynx complicating agranulocytic angina following sulfanilamide therapy.

2. Every practitioner should be familiar with the cardinal signs and symptoms of obstructive laryngeal dyspnea.

3. **Of all emergencies, obstructive laryngeal dyspnea is perhaps second to none. It often requires emergency relief and every physician should be equipped and capable to administer such relief.**

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441 Ocean Avenue.